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THE AMERICAN JOURNAL of Proctology

The Authoritative Journal of Proctology, Gastroenterology and Allied Subjects.

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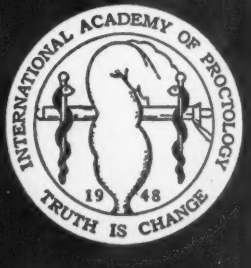
OFFICIAL PUBLICATION OF THE INTERNATIONAL ACADEMY OF PROCTOLOGY

Vermiform Appendix Lesions
Acute and Chronic Pancreatitis
Injuries to the Coccyx
Stress, Hormones and Inflammation
Acute Abdominal Pain
Proctologic Patient—Office Management
Editorial
Convention News
Therapeutic Abstract



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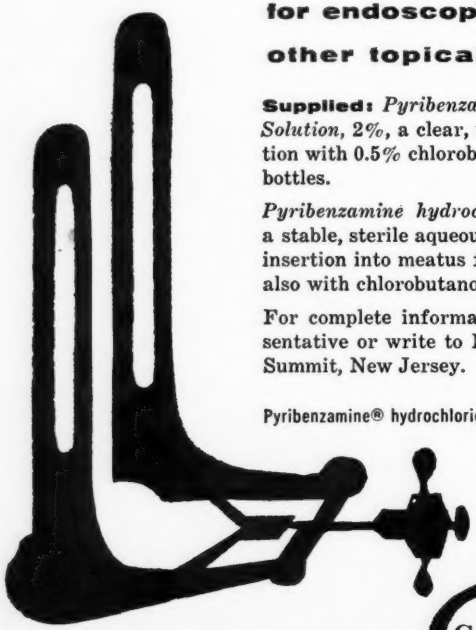

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As a special "bonus" to our readers, the Editors will increase the feature content of the next several issues. Of course, this will necessitate temporarily eliminating other editorial content, such as the Ambulatory Proctology section and the Atlas of Proctology. These sections will be resumed in subsequent issues.

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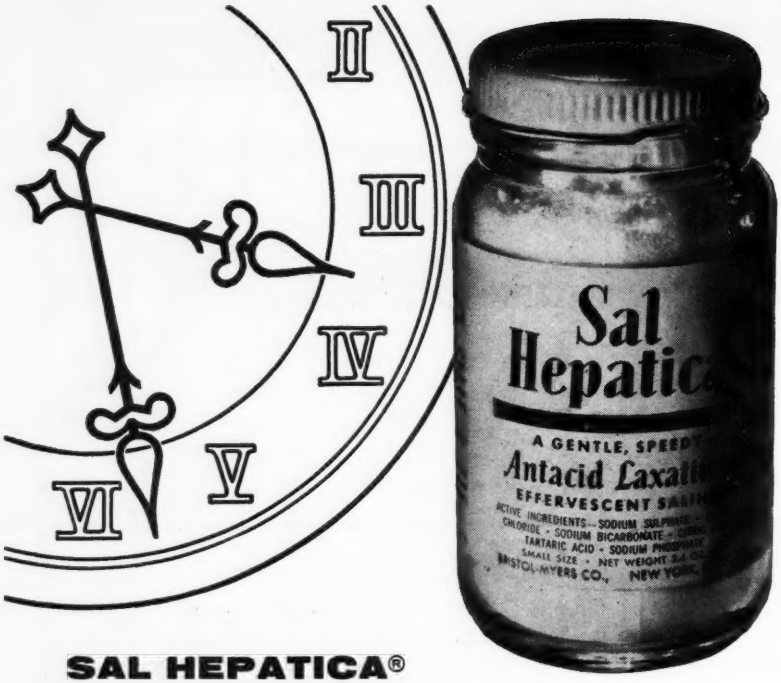

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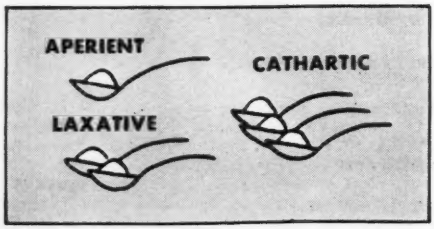

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1. The Physiological Basis of Medical Practice, 1945, p. 486.
2. New England J. Med. 235:80, July 18, 1946.

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The Authoritative Journal of Proctology, Gastroenterology and Allied Subjects.

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CONTRIBUTIONS Manuscripts for publication should be addressed to the Editor-in-Chief. Articles are accepted for publication with the understanding that they are contributed solely to this publication. When possible, two copies of the manuscript should be submitted. References should be compiled in accordance with the style used by the Quarterly Cumulative Index Medicus of the American Medical Association. The publishers will have up to four half-tones or line cuts made without expense to the authors. Reprints will be supplied the authors at cost. Original illustrations prepared by the publishers may be purchased by the authors it desired. Slides of these illustrations (standard size or 35 millimeter) can be supplied to the authors at cost.

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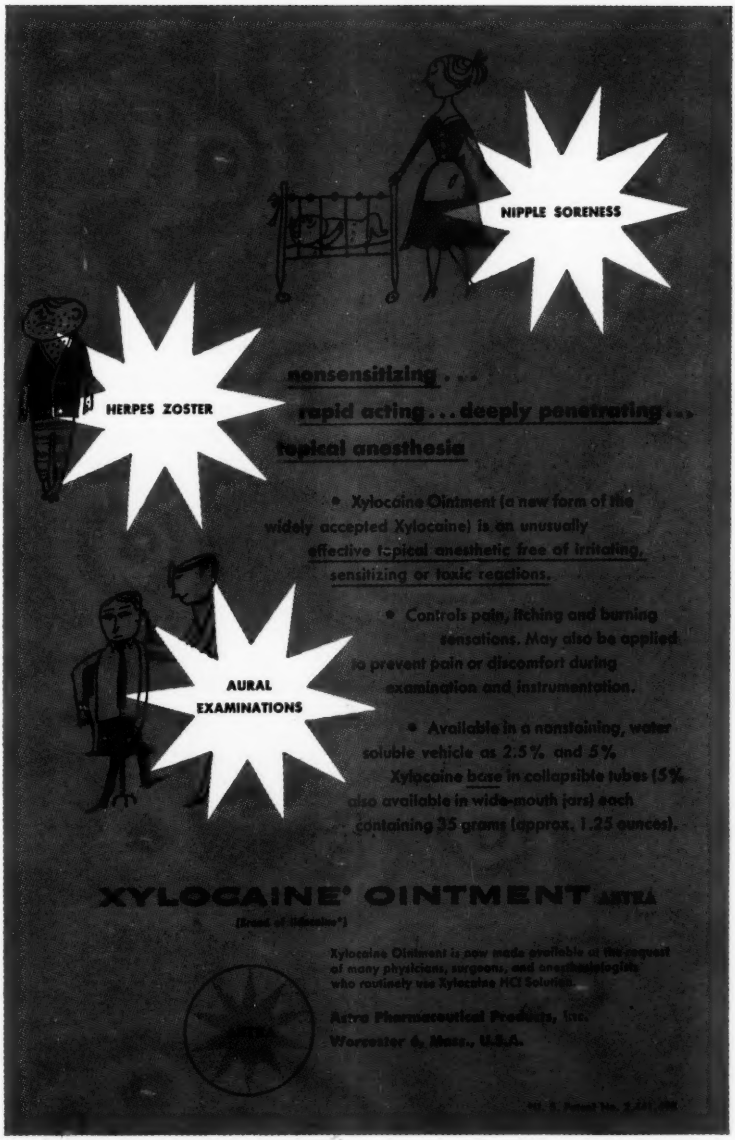
1. Ramsey, W.H.: Am. Pract. & Dig. Treat. 3:834.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LETTERS TO THE EDITOR

Dear Dr. Ruffin:

I very much enjoyed your excellent article on Severe or Fulminating Ulcerative Colitis in the February 1st issue of Modern Medicine. If you would care to abstract for the next issue of the American Journal of Proctology, we will be pleased to present this as an author's abstract.

Meanwhile, I should like additional information on the amount of cod liver-oil and the amount and nature of the nonabsorbable sulfonamide recommended for daily retention enemas. I would also appreciate clarification of the dosage of ACTH and the method of administration in these cases.

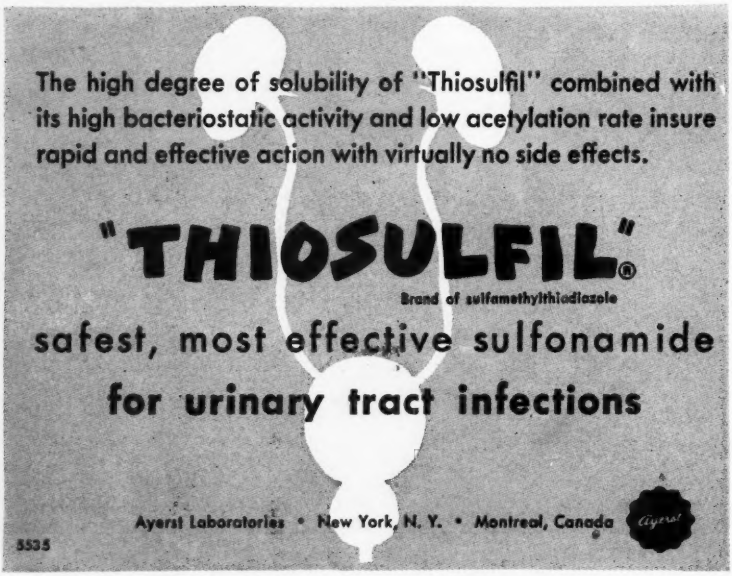
Sincerely,
ALFRED J. CANTOR, M.D. (Editor)

Dear Dr. Cantor:

Your letter of February 18th received. Thank you very much for your kind remarks in regard to my article. I shall be only too glad to prepare an abstract for the *American Journal of Proctology*. However, before doing so I will need to know the usual length of such abstracts.

In regard to the retention enemas, we usually use 100 cc. of cod liver oil, warmed to a comfortable temperature to which is added a teaspoonful of powdered sulfaguanidine. This is given as a retention enema once or twice daily, depending upon the severity of the disease. It has been found to be of

—Concluded on page 258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Unusual Lesions of the Vermiform Appendix

Based upon a Study of 24,326 Specimens

DONALD C. COLLINS, B.A., M.D., M.S. (Path.), M.S. (Surg.), Sc.D., F.A.C.S., F.I.C.S., F.A.C.C., F.A.C.G., F.I.A.P.
Hollywood, California

It is a genuine pleasure and a signal honor to be given the opportunity to present this subject before this Academy. You may correctly wonder just why this particular study is being given before a group of proctologists, and those particularly interested in diseases of the colon. Therefore, allow me to respectfully point out the following reasons for this presentation before you this morning:

1. The vermiform appendix represents the true apex of the cecum and the latter is the first portion of the large bowel.

2. Appendicitis is the commonest disease of the large intestine in America.

3. Because the appendix may occupy any position within the abdominal cavity, diseases of the appendix may mislead the physician into believing that he is confronted with an instance of diverticulitis or possibly an irritable colon syndrome. Such mistaken diagnoses are frequently accompanied by disastrous results.

4. Appendicitis occurring in either the very young or in the aged frequently present quite atypical signs and symptoms, and commonly traps the unwary into believing that he is dealing with an example of either enteritis or colitis. Disaster commonly ensues to that patient so erroneously treated.

5. Internal appendiceal fistula may produce very bizarre clinical pictures. One recent example was considered to be an instance of sigmoidal diverticulitis with a secondary perforation into the urinary bladder. All manner of local sigmoidoscopic treatments were a complete failure. Subsequent abdominal ex-

From the Departments of Surgery; The College of Medical Evangelists and The Hollywood-Presbyterian Hospital, Olmsted Memorial, Los Angeles, California.

Delivered before the Eighth Scientific Session, The International Academy of Proctology, The Palmer House, Chicago, Illinois, at 10:30 A.M., Saturday, April 10, 1954.

ploration revealed the true cause of that patient's illness, and appendectomy resulted in a prompt and brilliant cure!

6. Appendiceal disease may be responsible for severe rectal or perineal regional pain, and all manner of local proctoscopic therapy will fail to produce the desired relief of symptoms.

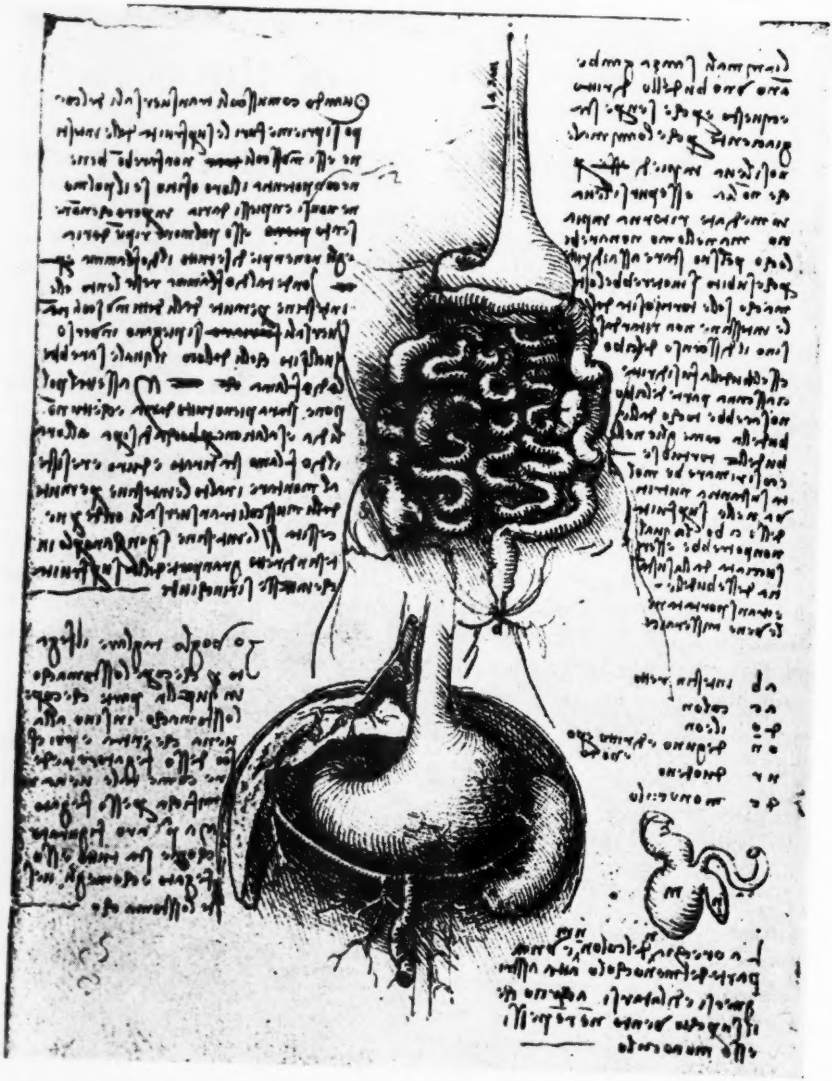

Fig. 1. Leonardo da Vinci (1472). First known drawing and description of human vermiform—appendix. (Courtesy of Dr. Elmer Belt of Los Angeles.)

Chart I

SOURCES OF DATA			
SOURCE OF MATERIAL	DIAGNOSIS	NUMBER	PER CENT OF 24,326
	A. ACUTE		
	1. Perforative	1,813	7.47
	2. Non-Perforative	5,117	21.05
	B. SUBACUTE	2,501	10.30
I. SURGICAL:	C. "CHRONIC"	4,145	16.99
(22,465 Cases or 92.35%.)	D. INFECTIOUS-GRANULOMATA	141	0.58
	E. PROPHYLACTIC-APPENDECTOMY-ONLY	8,748	35.96
II. POSTMORTEM:	VARIOUS	1,861	7.65
(1,861 Cases or 7.65%.)	(82% stated: "Not remarkable.")		
	GRAND TOTALS	24,326	100.00

7. Collections of pus in the cul de sac may be due to acute perforative appendicitis and not to left sided colonic disease.

Thus, with this very incomplete list of reasons, I hope that you will agree with me that the presentation of this study may prove of interest and benefit to both gastroenterologists and protologists.

The data of this work represents a summary of over twenty-eight years' constant study of the records of the hospitals with which I have been associated in San Francisco; Rochester, Minnesota; The United States Army Medical Department Hospitals during World War II; and Hollywood and Los Angeles, California. These thousands of appendices were removed by more than two hundred different surgeons. The pathologic diagnoses were made by well trained and competent pathologists in these respective hospitals. Several hundred thousand microscopic

Chart II

SPECIFIC DISEASES		
DISEASE	NUMBER	PER CENT OF 24,326
Tuberculosis, Secondary	62	0.255
Leprosy, Secondary	2	0.008
Syphilis, Secondary	49	0.201
Syphilis, Gumma	4	0.016
Actinomycosis, Secondary	10	0.041
Acute Exanthem	14	0.058
Acute & Chronic "C.U.C."	61	0.251
GRAND TOTALS	202	0.830

Chart III

PARASITIC DISEASES		
DISEASE	NUMBER	PER CENT OF 24,326
Amoebiasis	41	0.168
Malaria	36	0.148
Histoplasmosis	296	1.217
Echinococcus Cyst	8	0.033
Parasitic Worms in Lumen	94	0.386
GRAND TOTAL	475	1.952

glass slides were prepared while this data was being gathered.

For the purposes of brevity, the salient features of this presentation have been summarized in eleven charts. Bibliographic reference is made in these charts to more detailed descrip-

Chart IV

MISCELLANEOUS PATHOLOGIC CONDITIONS

DISEASE	NUMBER	PER CENT OF 24,326
Benign Lymphoma	97	0.399
Giant Lymphoid Hyperplasia	16	0.066
Melanosis Mucosae Appendicis-Vermiformis[4]	(4,958)	
(a) Severe	98	0.403
(b) Mild	4,860	19.980
Mucocele	174	0.715
Subserosal Cysts[37]	61	0.250
Diverticulae[†]	(474)	
(a) Acquired, Inflammatory	471	1.937
(b) True, Congenital	3	0.012
GRAND TOTAL	5,780	23.762

Chart V

FOREIGN BODIES IN THE LUMEN

TYPE OF FOREIGN BODY	NUMBER	PER CENT OF 24,326
Common Metal Pin	24	0.099
Metal Safety-Pin	3	0.012
Metal Screw	27	0.111
Nails & Metal Pieces	36	0.148
Fish & Animal Bones	51	0.210
Lead-Shot of Various Calibres	49	0.201
Wooden Splinters & Tooth-Picks	71	0.292
Fecoliths	(9,263)	
(a) Calcified	147	0.604
(b) Non-Calcified	9,116	37.476
GRAND TOTALS	9,524	39.153

Fig. 2. Appendix, operative specimen showing huge acquired diverticulum of middle-third.

Fig. 3. Roentgenogram, barium enema showing a diverticulum of vermiform appendix in proximal fourth, later proven by abdominal exploration and appendectomy.

Chart VI

BENIGN TUMORS

TUMOR	NUMBER	PER CENT OF 24,326
Mucosal Polyps[3]	17	0.070
Endometriosis[18]	16	0.066
Aberrant Pancreas[17]	19	0.078
Hyperplasia of A. & M. Plexi[4, 6]	516	2.121
Neuroma[13]	373	1.533
Ganglioneuroma[4]	64	0.263
Lipoma[4]	81	0.333
Fibroma	71	0.292
Adenomyoma[4]	12	0.049
Leiomyoma[4]	251	1.032
Leiofibromyoma	219	0.900
GRAND TOTALS	1,639	6.737

Chart VII

APPENDICEAL FISTULAE

TYPE OF FISTULA	NUMBER	PER CENT OF 24,326
A. EXTERNAL		
Appendix to Abdominal or Flank skin	27	0.111
B. INTERNAL		
Appendix to		
1. Terminal Ileum	21	0.086
2. Urinary Bladder	17	0.070
3. Sigmoid Colon	8	0.033
4. Rectum	5	0.021
5. Vagina	4	0.016
6. Fallopian Tube	5	0.021
7. Cecum	8	0.033
GRAND TOTALS	95	0.391

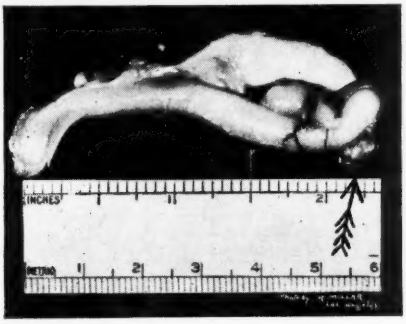

Fig. 4. Appendix, operative specimen. Arrow points to a carcinoid tumor at tip.

Chart VIII

MALIGNANT TUMORS

TYPE OF TUMOR	NUMBER	PER CENT OF 24,326
Malignant Mucosal Polyp[4, 17]	2	0.008
Carcinoid Tumors[4, 17]		
(a) Localized	71	0.292
(b) Metastasizing	7	0.029
Primary Adeno-Carcinoma[4]	18	0.074
Fibrosarcoma[17]	2	0.008
Neurosarcoma	3	0.012
Lymphosarcoma	2	0.008
Hodgkin's Disease	14	0.058
Pseudomyxoma Pertionaei, from the Appendix[4]	131	0.539
GRAND TOTALS	250	1.028

Chart IX

MISCELLANEOUS RARITIES

CONDITION	NUMBER	PER CENT OF 24,326
Congenital Agenesia[19]	1	0.004
True, Congenital Double Appendix	1	0.004
Post-Inflammatory Pseudo-Duplication[2]	1	0.004
Incomplete Rotation Appendix located in[4]		
1. L.L.Q.	4	0.016
2. L.U.Q.	11	0.045
3. R.U.Q.	892	3.667
4. Situs Transversus-Viscerum Abdominus	6	0.025
GRAND TOTALS	916	3.765

Chart X

OBLITERATION OF THE APPENDICEAL LUMEN

(Where Stated in 3,173 Specimens)

DEGREE OF LUMEN OBLITERATION	NUMBER	PERCENT OF 3,173
TOTAL[6, 11, 12, 15]	317	9.991
TWO-THIRDS	346	10.905
ONE-THIRD	491	15.474
NONE	2,019	63.629
GRAND TOTALS	3,173	99.999

Chart XI

POSITION OF THE APPENDIX IN THE ABDOMEN

(Where stated in 16,203 instances)

LOCATION OF APPENDIX	NUMBER	PER CENT OF 16,203
A. ANTERIOR TO THE CECUM[5, 3, 4, 8]	(12,074)	
1. Caudad, Towards the Pelvis	4,817	29.729
2. Mesial to Cecum	6,341	39.134
3. Lateral to Cecum	916	5.653
		74.516
B. RETRO-CECAL[9, 10, 11, 15, 16]	(4,129)	
1. Free	1,789	11.141
2. Fixed in Position	2,340	14.442
		25.583
GRAND TOTALS	16,203	100.099

tions of interesting pathologic details that have been presented in the literature since 1931. It is believed that this large number of appendices may have statistical value in the future study of others interested in the manifold diseases and abnormalities that plague the human vermiform appendix.

Bibliography

1. Collins, Donald C.: Historic Phases of Appendicitis. Ann. Surg. 94: 179-196 (August) 1931.
2. Collins, Donald C.: Pseudoduplication of the Vermiform Appendix. Surg. Clin. N. Amer. 11: 921-926 (August) 1931.
3. Collins, Donald C.: Adenomatous Polyps of the Vermiform Appendix. Surg. Clin. N. Amer. 12: 1063-1067 (August) 1932.
4. Collins, Donald C.: The Chronic Inflammatory and Obliterative Reactions of the Vermiform Appendix. Thesis. Post-Graduate School, University of Minnesota, June, 1932, 570 pp.
5. Collins, Donald C.: The Length and Position of the Vermiform Appendix. A Study of 4,680 Specimens. Ann. Surg. 96: 1044-1048 (December) 1932.
6. Collins, Donald C.: The Mechanism and Significance of Obliteration of the Vermiform Appendix, Thesis. Post-Graduate School, University of Minnesota, June, 1934, 92 pp.
7. Collins, Donald C.: Diverticula of the Vermiform Appendix. Ann. Surg. 104: 1001-1012 (December) 1936.
8. Collins, Donald C.: Treatment of Appendicitis. Am. Jour. Surg. 37: 440-445 (September) 1937.
9. Collins, Donald C.: Hematuria Associated with Acute Appendicitis. Urolog. & Cutan. Rev. 42: 22-24 (January) 1938.
10. Collins, Donald C.: Acute Retrocecal Appendicitis. Based upon 751 Instances. Arch. Surg. 36: 729-743 (May) 1938.
11. Collins, Donald C.: Etiological Factors in Acute Appendicitis. Surgery. 5: 267-270 (February) 1939.
12. Collins, Donald C.: Present Aspects of the Appendicitis Problem. Jour. Internat. Coll. Surgs. 4: 325-330 (August) 1941.
13. Collins, Donald C.: Present Concepts in the Management of Both Chronic and Acute Appendicitis. Jour. Internat. Coll. Surgs. 11: 154-169 (March-April) 1948.
14. Collins, Donald C.: The Grave Prognosis of Appendicitis in the Aged. Ann. Western Med. & Surg. 2: 193-205 (May) 1948.
15. Collins, Donald C.: Excision of Appendix. Modern Med 16:83 Only. (Oct.) 1948.
16. Collins, Donald C.: Suppurative Appendicitis. Modern Med. 17: 82 Only. (July 1) 1949.
17. Collins, Donald C.: Unusual Lesions of the Vermiform Appendix. Ann. Western Med. & Surg. 3: 384-387 (November) 1949.
18. Collins, Donald C.: Endometriosis of the Vermiform Appendix. Review of Literature with Addition of Nine New Instances. One of which Caused Severe Melena. A.M.A. Arch. Surg. 63: 617-622 (November) 1951.
19. Collins, Donald C.: Agenesis of the Vermiform Appendix. Amer. Jour. Surg. 82: 689-696 (December) 1951.

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Acute and Chronic Pancreatitis

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The manifold functions of the pancreas, and the highly varied visceral and vascular involvement of this gland, largely explain the lack of precise knowledge which still exists with respect to the etiology of its metabolic disorders. Diseases of the pancreas due to inflammatory reactions are especially a case in point, as will be clearly shown in the course of this summary.

The major manifestations of pancreatic disease due to inflammation may logically be grouped under two main headings—"acute" and "chronic" pancreatitis.

I. Acute Pancreatitis

1. Acute pancreatic ("glassy") edema, clinically described as mild acute or subacute pancreatitis.

2. Acute pancreatic necrosis (with many intermediate stages of severity), also known as acute hemorrhagic pancreatitis.

II. Chronic Pancreatitis

1. Chronic pancreatitis (constant symptoms).

2. Chronic relapsing or recurrent pancreatitis.

3. Pancreatic lithiasis and pancreatic calcinosis.

4. Cysts of inflammatory origin.

Acute Pancreatitis Acute inflammatory reactions of the pancreas, included under the heading of "acute pancreatitis", may occur pathologically in the form of acute pancreatic edema—a relatively mild form of the disease, or as acute pancreatic necrosis associated with severe clinical symptoms which may progress to suppuration and abscess formation.[1]

Etiology The etiology of these lesions remains obscure, although there is no dearth of theories based both on experimental data and on speculation. The great diversity of opinion existing in this respect among authorities is largely explained by the numerous variations which have been found to exist in the anatomical relationships between the biliary tract, the pancreatic duct, and

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the duodenum. However, on the basis of a comparative study of the extensive literature on the subject, Rienhoff and Pickrell[2] have assembled the six following etiological possibilities relative to pancreatitis:

1. Blocking of the duodenal orifice of the ampulla by a calculus, thus producing a "common channel" which permits bile to enter the pancreatic duct to infiltrate the pancreas and activate trypsinogen. Pancreatic tissue would then be digested and hemorrhage and fat necrosis would result.

2. Spacticity of the duodenal sphincter would produce the same result.

3. Infection of the pancreas from the gallbladder or from a distant focus. The infection might spread to the pancreas directly by extension from the duodenum by way of the pancreatic ducts, or by lymphatic or hematogenous spread. Some degree of obstruction of the pancreatic ducts is probably necessary for this development.

4. Complete obstruction of the pancreatic duct, from one cause or another may produce dilatation of the ducts and acini, and release pancreatic ferments with resulting hemorrhage and fat necrosis.

5. Hereditary: In some families several members are affected. Suggests congenital anomaly, or inborn error of metabolism.

6. Alcoholism: Population with high alcohol index has high incidence of pancreatitis. Perhaps many of the diagnoses of acute alcoholic gastritis may actually be mild attacks of pancreatitis.

Reflux of bile into the pancreas and subsequent rupture of the duct wall with escape of pancreatic juice and ferments into the interstitial pancreatic tissue are thus believed to be essential steps in the production of the inflammatory lesions of the pancreas. This reflux of bile into the substance of the gland is further believed to depend upon anatomical variation, gross interference with function, as by a stone, or functional upset in the workings of the sphincter of Oddi, due to emotional factors or to dietary indiscretions.[3] Alcoholic excess and biliary lithiasis have long been known as provocative accompaniments of attacks of acute pancreatitis.

It is obvious, in fact, that more than one type of obstructive factor may cause the escape of pancreatic juice from the ducts into the interstitial glandular tissue. Rich and Duff[4] have suggested that metaplasia of duct epithelium may be the common cause of obstruction. This hypothesis is not so well accepted, however, as their observations on the vascular changes which result from trypsin digestion. Their observation that the activity and concentration of trypsinogen or trypsin determines the degree of edema and the extent of vascular damage sounds very plausible.

Pathology Two forms of acute pancreatitis can be differentiated; namely, an acute interstitial, and an acute necrotic form which may be hemorrhagic, suppurative or gangrenous.

The interstitial or edematous form of acute pancreatitis is usually mild and the damage is quickly repaired. It is generally caused by arterial disease and is occasionally associated with cholecystitis.[5] The pancreas is enlarged, firm and edematous, and small foci of fat necrosis are present. There is hyperemia of the gland, and the interstitial tissue is infiltrated with polymorphonuclear leukocytes. A brown fluid exudes from the lesser peritoneal sac. This is

the more common form of the disease and responds well to the conservative treatment.[6]

In the acute necrotic form of the disease the gland is enlarged, firm, with soft friable areas of necrosis, or the entire organ may be converted into a mushy, friable, dark reddish-brown mass. If the enzymes are high in tryptic activity, hemorrhage results; if lipase predominates, diffuse small areas of fat necrosis are seen on the surface of the pancreas and in the surrounding mesenteric fat. The fat is broken down by the lipase into glycerin and fatty acids, and the latter combine with calcium to form insoluble soaps. Microscopically a specific vascular lesion is found, characterized by rapid necrosis of the walls of the arteries and veins, with hemorrhage resulting from the rupture of the necrotic vessels.[4,5] These several varieties of acute pancreatitis (interstitial or edematous, necrotic, hemorrhagic, suppurative, or abscess forming) may merge from one to the other.

Symptoms and Signs The nature and severity of the symptoms vary with the degree of involvement of the acinar tissue; they frequently lack diagnostic precision. However, there are certain subjective symptoms and findings which are fairly general. For example, the onset is usually sudden with acute excruciating pain in the epigastrium or upper abdomen, frequently radiating to the back, with tenderness over the upper abdomen and at times in the left costovertebral angle. Vomiting is usually severe, especially if the attack comes on after a heavy meal or alcoholic indulgence.

In the varieties associated with necrosis of vessels and interstitial hemorrhage, the symptoms of shock are marked and are most severe where necrosis of the gland exists. The pain is particularly refractory to average doses of narcotics, and is followed by repeated waves of nausea and vomiting and eventually obstipation and progressive abdominal distention.[1] Localized epigastric tenderness and rigidity become generalized and extreme as the disease develops. Fever is moderate and remittent. Demonstrable degrees of hemoconcentration and diminished blood volume are common and diagnostically helpful. Jaundice, probably due to hepatitis and obstruction of the common duct, is present in the severe forms of the disease. Ecchymotic discoloration[7] of the skin of the periumbilical and flank areas is indicative of retroperitoneal hemorrhage and occurs in approximately 10 per cent of the cases; it makes its appearance on the fourth or fifth day of the onset of the disease and is thus a late confirmatory sign.[8]

Laboratory Findings and Diagnosis The diagnosis is usually made on the basis of an acute, fulminating attack and on physical signs. The lesions which must be differentiated are those that require prompt surgical attention and those which can be treated by conservative measures. Perforated gastric and duodenal ulcers, acute cholecystitis, acute upper intestinal obstruction and acute appendicitis require prompt surgery. Coronary thrombosis and early pneumonia with diaphragmatic pleurisy fall into the second category. Other miscellaneous conditions which may produce abdominal symptoms are: uremia with nausea and vomiting, dissecting aneurysm, and non-specific gastroenteritis.

Laboratory findings include non-specific leucocytosis with an increase in

granulocytes and elevation of the hemoglobin and hematocrit values early in the disease.

The demonstration of a significant elevation of the amylase and lipase content of the blood and urine is diagnostic of acute pancreatitis. This increased serum amylase activity occurs rapidly, reaching a maximum concentration within 48 hours and then receding quickly to normal. The elevated findings in the urine persists for a longer period and is of more diagnostic help when the patient is not seen during the early stage of the attack. In a recent study by Edmondson et al.[9] of the serum amylase titer in 350 patients with acute abdominal disease, it was found that serum amylase levels above 500 units were almost exclusively present in patients with acute pancreatitis. Serum amylase concentrations in cases of perforation of the intestinal tract into the peritoneal cavity usually were below 250 units. It is also noted, however, by these authors, that the elevation of the serum amylase is not necessarily correlated with the severity of the disease or the prognosis, as is evident from their study of autopsy material.

The extent of pancreatic destruction is of course decisive. At one extreme is the almost completely necrotic, hemorrhagic gland, often with massive thrombosis of the pancreatic and splenic veins, which would obviously be incapable of producing an increased concentration of blood amylase. And the other extreme is the gland with only moderate necrosis of the acini and little hemorrhage, in which the ducts may contain clear secretion.

A difficult diagnostic problem is presented by some patients with severe hemorrhagic necrosis if they are first seen after the serum amylase has returned to normal. Fortunately, it is in this phase of the disease, as noted by Edmondson et al,[9] that hypocalcemia is of especial value in the diagnosis. In 27 patients with moderate to severe attacks of acute pancreatitis who were studied with respect to serum calcium and potassium changes, hypocalcemia was found in 19 or 70.1% of the cases. It appeared most often on the second to the fifth day of illness. Levels of calcium below 7.0 mg per cent are considered an unfavorable prognostic omen.

Hyperglycemia, glycosuria and diabetic tolerance curves occur in the devastating forms of the disease and must be looked for in all cases of acute pancreatitis. Hypoglycemia has likewise been observed.

Treatment Treatment of acute pancreatitis is non-operative if the clinical diagnosis can be established with reasonable certainty;[1] otherwise, an early laparotomy is preferable. Deliberate surgical intervention in the presence of recognized acute pancreatitis is reserved for those instances in which the inflammatory process goes on to suppuration or cyst formation. If the operation is performed during the acute phase of the disease, the procedure is limited to the insertion of a drain in the lesser peritonal sac, and the introduction of a T-tube into the common duct if the extrahepatic biliary tree is distended.

Conservative treatment is the rule for all cases of acute pancreatitis that have not progressed to the stage of suppuration and/or cyst or abscess formation. In fully developed pancreatic necrosis, for example, the prime therapeutic measures consist in gastro-intestinal aspiration and adequate replacement (includ-

ing calcium) of the blood volume and the electrolyte balance. Auxiliary measures, as recommended by J. K. Mowat,[10] include the following:

1. Administration of demerol for the relief of pain, without increasing the spasm of the sphincter of Oddi.
2. Administration of atropine, banthine or ephedrine to inhibit secretion of the acini.
3. Administration of papaverine to counteract reflex local arteriolar spasm, said to be a factor in the progression from glassy edema to massive necrosis.[11]
4. Paravertebral sympathetic and splanchnic block (likewise for relief of vascular spasm and of pain.[12]
5. Epidural procaine block.[13]
6. Administration of penicillin for control of secondary infection. Penicillin is the only antibiotic which appears high in concentration.
7. Use of insulin to combat hyperglycemia and glycosuria.

In the patients who fail to respond to conservative therapy and require surgery, the mortality is necessarily very high, varying from 10 to 30 per cent. Recovery from an attack may moreover be followed by a recurrence, especially if a calculous cholecystitis is present. There is also a small number of patients who have recovered from an acute pancreatitis who develop a chronic interstitial pancreatitis.[14]

Chronic Pancreatitis The term "chronic pancreatitis" is a rather indefinite term which covers a variety of lesions that are in general the end-result of a previous inflammatory process or of degenerative changes in the gland itself.[14] The disease is often difficult to diagnose with any degree of consistency. It is more prevalent in males than in females and has its highest frequency during the fifth and sixth decades.[15]

Etiology The disease may result from any cause of fibrosis of the pancreas, such as repeated attacks of acute pancreatitis; inflammatory disease of the biliary tract, stomach or dudenum; peptic ulcer penetrating into the pancreas; obstruction of the pancreatic ducts; tuberculosis; syphilis, or arteriosclerosis. Alcohol is believed to be an important etiologic factor in some instances.

Pathology Opie's classification of chronic pancreatitis into the "interlobar" and the "interacinar" forms is generally accepted. The essential pathology in both forms is an increase in fibrous connective tissue—the result of previous inflammatory or degenerative processes. The degenerative processes may be associated with areas of calcification and, when accompanied with calculus formation in the ducts, are indicative of previous acute hemorrhagic or necrotic disturbance. The extent of the fibrotic process through the organ, with destruction of the acinar cells and occlusions of the ducts, especially in the head of the pancreas, determines to a certain degree the extent of dysfunction in the digestion of fats and proteins.[14]

Symptoms and Signs The clinical manifestations of chronic pancreatitis depend on the nature of the associated organic disease, on the degree to which acinar and islet tissue are destroyed, and on whether or not obstructive jaundice is present. Where extensive degenerative changes and fibrosis have occurred, the findings are loss in body weight, frequent passage of foul, bulky,

fatty or greasy yellow or grayish stools that glisten because of the high fat content. Pain may be entirely lacking, but when present it is usually epigastric in location, with radiation through the back; it is aggravated by ingestion of fats or protein. Alcohol may likewise initiate an attack. Jaundice is not invariably present, but may be so if the head of the pancreas is involved by a fibrosing process in close proximity to the ampulla of Vater. When this condition prevails, not infrequently a diagnosis of carcinoma of the pancreas is made, not only by the clinician but by the surgeon in the operating room. The jaundice may be due, however, to the associated existence of biliary calculi. Diabetes mellitus with glycosuria may be present, but is not a constant accompaniment of the condition.[16]

Diagnosis A history of gradually increasing change in bowel habits together with the passage of fatty stools and loss of weight should suggest the possibility of chronic pancreatitis. The symptoms and signs listed above afford other important diagnostic evidence.

Laboratory indications are few. When chronic pancreatitis is accompanied by severe pain, an elevated concentration of serum amylase may be suspected. It must be remembered, however, that progressive destruction of the gland may significantly reduce the production of pancreatic amylase. In the absence of specific tests, determinations of pancreatic enzyme activity, either by microscopic examination of the stools or by study of the duodenal contents, may give a clue to the underlying process. The flow of pancreatic juice can be stimulated by the administration of secretion or cholinergic drugs, or by the ingestion of a fatty meal. Prostigmine increases by 33% the pancreatic secretion by stimulating the parasympathetics. A film of the upper abdomen is frequently of great help in demonstrating disseminated calcification of the pancreas, or in suggesting the presence of a pancreatic calculus.

Treatment When severe pain is not a prominent feature, the essential treatment consists in the use of potent pancreatic extracts. Preparations are now available which contain adequate amounts of potent proteolytic, lipolytic and amylolytic enzymes which may be administered with the meals. The addition of an emulsifying agent, such as "Tween-80," may increase fat absorption, which also may be aided by the addition of choline or methionine. Ingestion of protein hydrolysates may help in maintaining a positive nitrogen balance. Sufficiency of vitamins and mineral intake should be assured. If diabetes is present, it must be treated with insulin. It is rarely possible, however, to correct the deficiency in pancreatic enzyme activity completely.

In instances in which pain is an important feature, the therapeutic problem is a complicated one, for these patients tend to become addicted to narcotics and excessive use of alcohol to relieve their discomfort. When the problem of intractable pain is present, surgical intervention is frequently necessary.

Two major surgical approaches may be considered; namely, total pancreatectomy and splanchnicectomy. Such a radical procedure as pancreatectomy is justified only in extreme cases and should be avoided because of its undesirable side-effects. The resultant medicinal and dietary measures necessary to maintain nutrition are very complicated, and the diabetes which has been pro-

duced must of course be taken care of.

On the other hand, adequate symptomatic relief may be obtained by splanchnicectomy. A left splanchnicectomy occasionally gives complete relief from pain; but in some instances, bilateral section of the sensory pathways may have to be performed.[16] Finally, when manifestations of obstructive jaundice are present, it may be necessary to proceed surgically by doing a cholecystenterostomy or cholecystogastrostomy.

Chronic Relapsing (or Recurrent) Pancreatitis was first clearly differentiated from other pancreatic disease and described as a clinical entity by Comfort, Gambill and Baggenstoss of the Mayo Clinic.[17] The term was applied to a symptom complex, characterized by recurrent bouts of upper abdominal pain associated with or followed by demonstrable disturbances in function of both acinar and islet tissue. The disease often progresses to a chronic state, complicated by fibrosis and calcification, leading to persistent pain, steatorrhea and diabetes. It is apparently a relatively rare disease[18] and its etiology is not definitely known.

Pathologically, the disease appears to be the result of recurrent attacks of varying degrees of intensity of acute pancreatitis, with the pancreas revealing evidence of edema, hemorrhagic necrosis, cystic degeneration, fibrosis, atrophy or calcification. The disease may precede or follow the development of cholelithiasis.[18]

Symptoms and Diagnosis Pain is the most characteristic symptom. An acute attack of severe pain was the first sign of the disease in 14 of 29 cases of recurrent pancreatitis reported recently from the Mayo Clinic. Pain in the acute episodes of the disease may be sudden in onset and termination, but it often begins mildly, gradually increases in severity until a plateau is reached, and then gradually subsides in a period of hours or days.[17] The pain tends to be steady rather than colicky, and has been variously described as burning, sharp, stabbing, cramping, pressing or aching.

The most characteristic feature of the acute attack is its prolonged duration, for it seldom terminates before the lapse of 2 to 14 days, and sometimes longer. Unlike uncomplicated biliary colic, one injection of morphine does not usually terminate these attacks of pancreatitis.[18]

The primary site of the pain may be in the right, middle or left part of the epigastrium or, rarely, in the lower thoracic portion of the back. The pain may radiate to various parts of the abdomen, thorax, and thoracolumbar region of the back. Pain which starts in the epigastrium and shifts to the left upper portion of the abdomen and the left side of the back is highly suggestive of pancreatitis.

Acute exacerbations of the disease were followed by certain permanent sequelae, including diabetes, steatorrhea and calcification, which were recognizable in the interval between attacks and were of diagnostic value, in about one-half of 29 patients with relapsing pancreatitis which were studied at the Mayo Clinic by Comfort and associates. In the other half of the cases, it was not possible to demonstrate pancreatitis by physical or laboratory methods applied during the interval between attacks. On the other hand, both surgeon and pathologist were able to demonstrate the existence of characteristic pathologic changes. In this latter series

of patients, destruction of the pancreas had not progressed to the stage of permanent sequelae. In such instances, a tentative diagnosis of relapsing pancreatitis may be made only after the exclusion of all other causes of abdominal pain, and on the basis of recurrent episodes of upper abdominal pain. It is in this type of case that an accurate description of the painful episode, including the type of onset and conclusion of the attack, the character of the pain, its location, radiation and duration, is of prime importance in making a presumptive diagnosis of recurrent pancreatitis.

If, on the other hand, destruction of the pancreas has progressed to the point where it becomes demonstrable during the intervals between the attacks, the diagnosis may be based on the following criteria: diminution of secretion of enzymes, bicarbonate, and volume of the pancreatic juice; excess of fat and of nitrogen in the feces; diabetes; deposition of calcium in the pancreas, and/or the formation of pseudocysts.[18]

Deposition of calcium was demonstrated roentgenologically in 13 of the 29 cases reported by the Mayo Clinic. It was demonstrated as early as the first and as late as the 19th year after the initial attack; it is deposited as areas of calcification in the parenchyma, or as stones in the ducts.[17]

Anorexia, nausea and vomiting and diarrhea are among the common miscellaneous sequelae which account for the progressive deterioration of the nutritional status of these patients. Anemia is common and hypochromic in type. The acute, painful attacks may diminish in severity and frequency, but they rarely cease completely before or after extensive damage has been done to the pancreas.

Death may result from the diseased pancreas itself, that is, from acute necrosis, gross gastro-intestinal hemorrhage, pyelophlebitis or abscess of the pancreas. More often, however, the fatal ending is due to unrelated causes, such as carcinoma, cerebral vascular accidents, or intercurrent infections.[17]

Treatment Medical treatment is mainly palliative and is especially directed to the relief of pain. During an acute attack, opiates, intravenous administration of fluids, decompression of the gastro-intestinal tract, and accepted methods to cope with shock, are employed.

During the chronic phases, a bland diet and abstinence from alcohol are basic requirements; diabetes is controlled; external pancreatic insufficiency and the resulting steatorrhea and azotorrhea are treated by a high caloric, high protein, high carbohydrate, low fat diet, by substitution therapy in the form of enteric-coated pancreatin tablets, or both measures.

Since pain is the most challenging problem of this disease, surgical treatment alone (e.g., unilateral or bilateral extensive splanchnicectomy) appears to offer prolonged relief from the painful attacks, although such relief is attained in only about 50 per cent of the cases subjected to surgery.[17] If no associated abdominal disease is demonstrable, medical measures should in general be given a reasonable trial first.

Bibliography

1. Cattell, R. B. and Warren, K. W.: Surgery of the Pancreas. Practitioner (London), 162: 188-195, 1949.

2. Rienhoff, W. F., Jr. and Pickrell, K. L.: Pancreatitis: Anatomic Study of Pancreatic and

Extrahepatic Biliary Systems. Arch. Surg., 51:205-219, 1946.

3. Doubilet, H. and Mulholland, J. H.: Recurrent Acute Pancreatitis. Observations on Etiology and Treatment. Ann. Surg., 128:609-636, Oct. 1948.

4. Rich, A. R. and Duff, G. L.: Experimental and Pathological Studies on Pathogenesis of Acute Hemorrhagic Pancreatitis. Bull. Johns Hopkins Hosp. 58:212-259, 1936.

5. Kantor, J. L. and Kasich, A. M.: Handbook of Digestive Diseases, 2nd Ed., p. 658, Mosby, 1949.

6. Elman, Robert: Surgery in Acute Pancreatitis. Gastroenterol. 7:656-664, 1946.

7. Turner, G. G.: Local Discoloration of the Abdominal Walls as a Sign of Acute Pancreatitis. Brit. J. Surg., 7:394, Jan. 1920.

8. Lipp, W. F.: Acute Pancreatitis. Report of a Case Showing Serum Calcium Changes as Well as a Gray-Turner Sign. Gastroenterol. 7:569-573, 1946.

9. Edmondson, H. A., Berne, F. J., Homann, R. E. and Ertman, M.: Calcium, Potassium, Magnesium and Amylace Disturbances in Acute Pancreatitis. Am. J. Med., 12:34-42, Jan. 1952.

10. Mowat, J. K.: (Brisbane) Some Observations on Pancreatic Pain. Med. J. Australia, 38:824-828. June 1951.

11. Popper, H. L., Necheles, H., and Russel, K. C.: Transition of Pancreatic Edema into Pancreatic Necrosis. Surg. Gyne. & Ost., 87:79-82, 1948.

12. Warren, K. W.: Acute and Chronic Pancreatic Pain. Surg. Clin. No. America, 30:873-883, June 1950.

13. Orr, R. B. and Warren, K. W.: Continuous Epidural Analgesia in Acute Pancreatitis. Lahey Clinic Bulletin, 6:204-210, 1950.

14. Whipple, A. O.: Pancreatitis (Acute and Chronic), R. L. Cecil, Textbook of Medicine, 7th Ed., Saunders, 1947, 893-897.

15. T. R. Harrison, Ed., Principles of Internal Medicine, Blakiston, 1950, p. 1475.

16. Musser-Wohl, Eds., Internal Medicine, Its Theory and Practice, 5th Ed., Lea & Febiger, 1951, p. 1315-1317.

17. Comfort, M. W., Gambill, E. E. and Bagenstoss, A. H.: Chronic Relapsing Pancreatitis. A Study of 29 Cases without Associated Disease of the Biliary or Gastrointestinal Tracts Gastroenterol., 6:239-285, 337-408, 1946.

18. Gambill, E. E. Baggenstoss, A. H. and Comfort, M. W.: Symposium on Chronic Relapsing Pancreatitis. Proc. Staff Meet. Mayo Clinic, 22:537-552, Nov. 26, 1947.

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Injuries to the Coccyx*

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Opinions regarding injuries to the coccyx are somewhat controversial. Many surgeons say that there are few injuries to the coccyx; some find that they occur occasionally; and my own experience confirms that of others who report that they occur frequently. Injuries to the coccyx fall into four categories: (1) sprain of the ligament or muscle; (2) fractures, both open and closed; (3) dislocations, with and without fracture, and (4) arthritis.

A word about the type of structure may be in order: The coccyx is a suspended articulated series of small bones, ligamentous attachments, and muscle attachments. It is located at the end of the spinal column. There are nerves known as the sacrococcygeal plexus which are importantly involved in injuries to the coccyx. Coccygodynia is the term applied to a painful coccyx or to an injured coccyx, painful as a result of the injury. There is a normal position for the male, which is a gentle forward curve. Any deviation from this normal curve is abnormal. There is no normal position of the coccyx in the female.

Sprains

Occurrence. Sprains result from falls or as an injury incurred while performing, such as happens to dancers in whom trauma has a high incidence because of the wide spread of the limbs, which may stretch or tear the ligamentous structures. Softening of the ligamentous structures during pregnancy predisposes to trauma or injury in this area; consequently, childbirth frequently causes a sprain of the coccyx, and, indeed, even a fracture or dislocation. Pain in the coccyx often follows surgery, such as abdominal-perineal resection or any type of resection or repair of the perineum. It may likewise follow too vigorous manipulations during rectal examinations. Proctoscopic examination may produce pressure on the ligamentous structures and thus precipitate a sprain. Horseback riding and bicycle riding or any occupation calling

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for constant pressure and trauma in a sitting position, which is a relaxed position, will also cause a sprain in this area.

Treatment Hot sitz baths, sitting in a tub of hot Epsom salt water for 6 or 8 minutes three times a day is beneficial. Opium and belladonna suppositories, inserted each evening at bedtime, are also beneficial. The bowels should be kept loose, and an attempt made to eliminate coughing or straining. All irritation, including that incurred during normal occupation, should be discontinued until the pain leaves. The use of a tight-fitting girdle or corset assists women in immobilization of this area. Men may also use a two-way stretch or an elastic girdle to aid in immobilization. An air cushion is helpful because a hard chair as well as an extremely soft chair may aggravate the condition.

Fig. 1. Deformity in a female which is normal, showing a marked displacement that occurs without symptoms.

It must be emphasized that injuries to the coccyx require a long period of rest and recuperation. There is no shortcut to recovery. It takes from 6 to 12 weeks before pain is relieved, regardless of the program of treatment.

Fractures may be open or closed.

Open Fractures Fortunately the open fracture is not encountered too often. When it does occur, complete débridement is essential, and one must make sure that there are no perforations in the descending colon. If perforations have taken place in the descending colon, a resection with a colostomy is preferable to closure of the descending colon with the attendant risk of infection. At the time of debridement, it is usually necessary to remove fragments and sometimes even the entire coccyx in order to complete the repair.

Closed Fractures Diagnosis of a closed fractured coccyx bone is difficult. Confirmation by the roentgenologist as to whether there is or is not a fracture is not always definitive. On rectal examination the area of the coccyx will be tender and usually freely movable, independently of the main portion of the coccyx. Manipulations of this fracture require the utmost care. Patients should be kept in bed.

Treatment calls for hot sitz baths, opium and belladonna suppositories, warm enemas to make sure that stools are not firm but are loose, and relief of any other condition that might induce muscle contracture at the rectum. Complete rest is required for at least 3 weeks. Support is then provided by a pelvic girdle, a belt, or some type of strapping that will hold the buttocks together, and to some extent immobilize the fractured coccyx.

Fig. 2. This shows a deformity of the coccyx in a female, which is normal.

Any displacement found on rectal examination must be corrected. Patients are placed on a non-gaseous diet so as to prevent distention. In this group of fracture injuries there is often ecchymosis and swelling around the anus, which is extremely tender, particularly over the coccygeal area. If the distal fragment has been completely separated or displaced, it should be pulled forward by the anococcygeal and levator ani muscles.

It is strange that so little has been written on a common condition encountered so frequently. It is interesting that in *Medical Records* of December 17, 1887 Jolly reported a unique case of the escape of the lower segment of the coccyx through the anus ten days after delivery, the bone apparently having been broken at that time.

Dislocations Complete dislocations may be forward or anterior, backward or posterior, or lateral. Dislocations forward are usually caused by violence or by a fall upon the buttock with the patient in a sitting position. This naturally forces the lower segments of coccyx forward, causing a severe sudden pain after the change of position. Many patients faint because of the severity of the pain. There is also pain on defecation and frequently an urgency to urinate. Pain usually radiates down the thighs and sometimes over the trunk. The patient is unable to sit up, and the slightest movement may greatly increase the pain. Coughing and sneezing, and sometimes even an act of inspiration, increases the pain in the coccygeal area. Internal examination reveals a forward displacement of the bone. External examination may show ecchymosis and swelling over the coccyx and a slight displacement of the bone forward.

Internal examination must be done under anaesthesia. A general anaesthetic is preferable to local anaesthetics. On internal examination the finger

Fig. 3. This shows a deformity of the coccyx in a male with a coccygodynia.

should be hooked over the projecting end of the coccyx and drawn back on the fragments. Frequently a snap will be heard and felt as the reduction is effected. Instantaneous relief of all symptoms may follow. It must be emphasized, however, that there is a strong tendency to recurrence, so that it may be necessary to manipulate or reduce the dislocation several times.

The backward dislocation is usually found in obstetrical cases, in which, because of increased ligamentous relaxation, a dislocation occurs or a fracture-dislocation may take place as a result of previous injury to the coccyx.

Lateral dislocations are not common, but they do happen and their possibility should not be overlooked. There may be a combination of a forward-lateral or a backward-lateral displacement. This type of lesion will frequently cause unilateral pain instead of a central or a bilateral pain down the inner sides of both thighs. There is, moreover, often a disturbance of sensation resulting from an injury to the sacral plexus.

In those cases in which a fracture complicates the dislocation and there is extensive displacement, removal of the coccyx seems to be the only remedy, because it is impossible to splint this fracture in the correct position. An attempt to replace may be complicated by severe injury to the lower bowel or to the rectum. Exostosis following a fracture or a fracture-dislocation is a possible complication in this area which is often overlooked. It can become large, is usually painful, and may necessitate removal by an open operation.

Arthritis Osteo-arthritis may develop at the sacro-coccygeal junction or at any of the articulations of the coccyx. Osteo-arthritic changes result in degeneration of the articular cartilage with a roughening at the articulation, usually accompanied by a chronic or constant pain. Patients not uncommonly become narcotic addicts because of the severe constant pain and the inability to sit for any length of time. Many of these patients have mental disturbances.

Because of the constant irritation, sacro-coccygeal ligamentous structures

Fig. 4. This shows a healing fracture just above the junction of the coccyx causing a marked coccygodynia.

are tight. A tight band is found on rectal examination which is usually unilateral, although it may be bilateral. Ironing or relaxation of this band by massage to loosen it often gives relief, although it is painful at the time. Such manipulation may require an anesthetic; however, if it can be done with-

out an anesthetic so as to induce frequent relaxing of this ligament by rectal manipulation, relief will be greater. Hydrocortone injections, hot sitz baths, tight pelvic girdles, daily warm enemas, and suppositories containing opium are alleviating adjuncts to treatment.

Injection and Surgical Treatment for All Types

Novocaine and Alcohol Injections It has been popular to inject the sacro-coccygeal articulation with novocaine. If the novocaine helps, a long-lasting novocaine should be used, and repeated injection is warranted. Some observers believe that if relief is obtained from the local anesthetic, then alcohol injection is indicated.

An instance in my experience warns that the use of absolute alcohol is not without danger in some cases: I had assured an apprehensive patient who had received relief from the novocaine injection, that open operation was not to be performed but that it was necessary that she be anesthetized in order to inject the area properly. Accordingly 0.5 cc. of absolute alcohol was injected into the sacro-coccygeal articulation. Upon awakening the patient experienced severe pain, for which narcotics and opium suppositories were administered. Two days later there was complete relief, but three weeks later the patient informed me that there was an open area over the region of the injection, from which she assumed that I had, after all, performed surgery.

On examination I found that the entire area for about 1½ inches by ½ inch was indeed wide open; that there was a marked slough over an area of about 1½ inches by ½ inch which finally healed, leaving a large scar. Although the patient was relieved of pain.

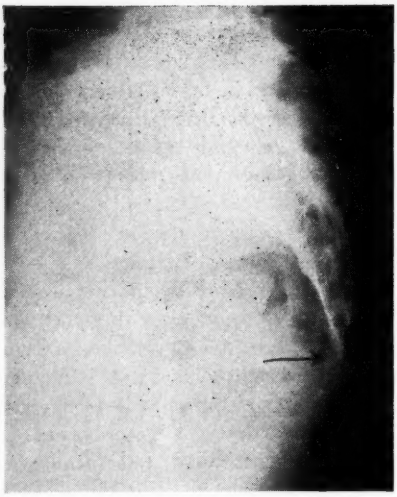

Fig. 5. This demonstrates a fracture of the sacrum just above the coccyx, with displacement causing a painful coccyx.

the situation was embarrassing, because I had promised that there would be no surgery.

Operative Procedure Thorough preparation should include administration of antibiotics and sodium succinate, so that if a perforation should occur, at least the bowel will be sterile.

An incision is made over the coccyx with the operator's finger in the rectum so as to guide the incision in order that the rectum will not be perforated. A subperiosteal dissection is accomplished with care not to injure the ligamentous attachments. The entire coccyx is removed.

It is preferable to begin at the sacrum and dissect distally than to advance from the distal to the proximal. A better result is obtained when the proximal segment can be retained. If, however, there are severe osteo-arthritic changes at the sacro-coccygeal area, then the

coccyx should be removed at the sacrum, and the sacrum should be beveled so as not to cause a posterior ledge or protrusion. It is worth repeating that if it is possible to save the proximal fragment and remove the remaining segments, it is highly preferable to do so. It is important to closely approximate the ligamentous structures that have been dissected away subperiosteally from the segments and to suture the ligamentous structures in a tight or imbricated position so as to form an accurate reattachment. The skin is then closed with silk and a pad applied.

Postoperative Care Patients must be kept comfortable. The diet must not include any gaseous food. Stools must be kept loose so that there will be no straining on bowel movement. Straining as well as coughing causes a contraction in the rectal area and increases the amount of pain. Sedation by mouth and opium suppositories are again utilized in order to give the patient relief.

Postoperative Regrowth It is not unusual to find, on roentgenologic examination made 6 months and a year later, a regrowth of some of the bone where the ligaments were separated subperiosteally, thus making the ligamentous attachments a firm support. This is important in preventing a further recurrence of the pain or a coccygodynia.

Course The accompanying illustrations represent the various positions that can be normal for patients without causing serious discomfort. The fracture of the lower portion of the sacrum causes a reflex pain in the coccyx. It is only in the area of the coccyx that pain is felt. Manipulation of the coccyx aggravates the pain. Frequently an excess amount of callus is formed after a fracture of the sacrum which interferes with the sacral plexus, causing a referred pain in the coccygeal area and resulting in a coccygodynia. For this reason it is important to keep patients quiet for at least 3 or 4 weeks. Frequent rectal examinations are necessary to determine the status of growth. Patients must be impressed and forewarned that as in all fractures, dislocations, or sprains of the coccyx, this serious injury requires at least 4 months before relief is obtained.

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Stress, Hormones and Inflammation

HANS SELYE, M.D., Ph.D., D.Sc., F.R.S. (C.), F.I.C.S. (Hon.)*
Montreal, Canada

In 1953 when I was honored by this Academy with the invitation of presenting before you a synopsis of our work, I chose as my title "Stress and Inflammation,"[1] laying particular emphasis upon the role played by hormones in regulating the "inflammatory potential," that is, the ability of the body to respond to injury with inflammation.

A brief synopsis of the whole "stress theory" has been the topic of a series of lectures which were subsequently published in the form of a booklet.[2] A more advanced discussion dealing with the role of systemic or local stress in the production and treatment of gastrointestinal disturbances will be found in the original monograph "Stress"[3] and in the four "Annual Reports on Stress"[4,5,6,7] which were published during the last four years. I shall therefore limit myself here to a description of certain fundamental new observations which strengthen the hypothesis I outlined before you at your Meeting of May 30th, 1953.

You will recall that, at that time, we discussed indirect experimental evidence suggesting that the ability of tissues to respond to injury with inflammation is largely dependent upon the activity of so-called antiphlogistic corticoids (e.g. hydrocortisone or cortisol**) and prophlogistic corticoids (e.g. desoxycorticosterone acetate, DCA). That cortisone and cortisol are in fact inhibitors, while DCA is a stimulator of inflammation, has received ample confirmation. It has also been demonstrated beyond doubt that the human adrenal cortex does actually produce significant amounts of such anti-inflammatory hormones. On the other hand, several investigators expressed doubt that DCA is a naturally occurring hormone and that the adrenal can under any circumstances actually stimulate inflammatory processes through hormonal means.

It is of particular importance, therefore, that recently a new DCA-like substance, "aldosterone," has been found

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** In agreement with the suggestion made by Shoppee,[8] the term cortisol is used here in preference to "hydrocortisone," since it eliminates confusion with 4,5-dihydrocortisone and obviates the possible implication that "hydrocortisone" is to be regarded primarily as a mere derivative of cortisone.

Presented at Seventh Annual Teaching Seminar, International Academy of Proctology, New York City, March 1955.

not only in the adrenal but also in the circulating blood and urine of man.[9] This new hormone is about 40 times as active as DCA as regards its effect upon mineral metabolism, but it still had to be proven whether it resembles DCA in antagonizing cortisone and cortisol, as well as in stimulating inflammation.

Extensive investigations, using the granuloma-pouch technique [which I had described before you two years ago[1]] have shown that, at the dose-level of 50 μg/day, aldosterone (generously supplied by Dr. A. Wettstein of Ciba, Basle) and DCA are about equally potent in antagonizing the actions of cortisol upon the body-weight and the development of the lymphatic organs. In addition, it has been found that the new natural mineralo-corticoid also resembles DCA in that it inhibits the atrophy normally induced by cortisol in the skin, bones and muscles.

Although aldosterone was shown by others to possess an unusually high mineralo-corticoid activity, its anti-cortisol effect would thus appear to be only about the same as that of DCA. It is noteworthy, however, that even so the natural mineralo-corticoid (aldosterone) inhibits all the above mentioned morphologic action of the natural glucocorticoid (cortisol) in a proportion of 1:8.

Thus, the concept according to which *a balance between two opposing naturally secreted corticoids can regulate the course of various biologic phenomena, including inflammation, has now been proven by direct experimental observations, using corticoids whose presence in the circulating blood had been demonstrated beyond doubt.*[10]

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References

1. Selye, H.: Stress and Inflammation. Am. J. Proctol. 4, 229, 1953.
2. Selye, H.: The Story of the Adaptation Syndrome. Acta, Inc., Med. Publ., Montreal, 1952.
3. Selye, H.: Stress. The Physiology and Pathology of Exposure to Stress. Acta Inc., Med. Publ., Montreal, 1950.
4. Selye, H.: First Annual Report on Stress. Acta Inc., Med. Publ., Montreal, 1951.
5. Selye, H. and A. Horava: Second Annual Report on Stress. Acta Inc., Med. Publ., Montreal, 1952.
6. Selye, H. and A. Horava: Third Annual Report on Stress. Acta Inc., Med. Publ., Montreal, 1953.
7. Selye, H. and G. Heuser: Fourth Annual Report on Stress. Acta Inc., Med. Publ., Montreal, 1954.
8. Shoppee, C. W.: The chemistry of cortisone. In: Annual Review of Biochemistry, 22, 261. Ann. Reviews, Inc., Stanford, Cal., 1953.
9. Simpson, S. A. and J. F. Tait: Aldosterone and its possible role in the general adaptation syndrome. In: H. Selye and G. Heuser: Fourth Annual Report on Stress, p. 206, Acta Inc., Med. Publ., Montreal, 1954.
10. Selye, H.: Addendum on the anti-cortisol effect of aldosterone. In: H. Selye and G. Heuser: Fourth Annual Report on Stress, p. 552, Acta Inc., Med. Publ., Montreal, 1954.

659 Milton Street

Acute Abdominal Pain

A Result of Dissecting Aortic Aneurysm*

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PART I

Abdominal pain referable to the bowel has often caused physicians grave concern. As a result, a voluminous literature has accumulated stressing points in diagnosis, advocating regimens of treatment and elaborately detailing surgical techniques. Within the last decade, dissecting aneurysms of the aorta have increased in frequency, and now deserve consideration in the differential diagnosis of extrinsic bowel conditions. Still more important is recognition of the fact that surgeons called upon to operate for supposed bowel obstructions or vascular impairment to the bowel, may have to deal with a dissecting aortic aneurysm. Established blood vessel banks are functioning in most large population centers and in numerous institutions where vascular surgery is far past the experimental stage. The potentialities for life-saving procedures by team-work on the part of abdominal and vascular surgeons are suggested by the following patients. They all had pain referable to the bowel, at least as a major part of their symptomatology. They all died of dissecting aortic aneurysm.

Case Reports

Case I—The first patient, B. F., a Jesuit priest 67 years of age, was brought by ambulance to the Loretto Hospital on October 26, 1950. He complained of severe abdominal pain, which was most intense in the left lower quadrant. The pain was not relieved by an enema or by 1/6 grain (0.01 gm.) of morphine sulfate given hypodermically. A second similar injection of morphine given three hours later induced a brief fitful sleep. The patient awakened with pain in the abdomen and back and moaned and tossed in agony. He had been in the hospital only six hours in his terminal stage and was thought to have a mesenteric thrombosis.

The blood pressure was 190/110 on several readings and was in marked contrast to his usual pressure of 124/80. The lower pressure had been recorded

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on numerous occasions in the preceding six years and even while the patient had been hospitalized for coronary insufficiency and myocardial infarction.

The pain in the abdomen was difficult to evaluate, because of a history of so-called colitis. Various episodes of this condition had been self-medicated with cathartics and alkaline powder for many years. An especially severe abdominal pain five days before death had been so treated. This was at a time, in the last two weeks of October, 1950, when the patient had a variety of other symptoms. First hemiparesis occurred and was associated with dysarthria. The conditions developed over several days and resulted only in a minor disability in movement. They had improved to the point where the patient had resumed his duties just prior to his death.

A review of his personal history disclosed "rheumatic trouble" at six years of age, but details were lacking. There was hospitalization in 1939 for repair of a ruptured left quadriceps femoris muscle. This was incurred fourteen months previously in a baseball game. At this time the heart had a normal rate and rhythm and all laboratory tests showed normal blood and urine.

Despite the good condition of the heart in 1939, the patient had a severe anginal attack in 1940. His condition improved after a ten day stay in the hospital. The electrocardiogram showed some inversion of the T wave in lead 1. No repetition of the attack occurred for six years, and in the interval the patient resumed his duties as a religious instructor. His pulse was good, and his blood pressure was from 115 to 125 systolic and 70 to 80 diastolic.

In February, 1946, the patient had two attacks of angina pectoris. These were so severe that he recorded their occurrence distinctly at 10 a.m. and 1 p.m. The pain was in the chest at the level of the heart and radiated to each shoulder. Glyceryl trinitrate therapy relieved the pain. The electrocardiogram showed low-voltage QRS complexes, suggesting diffuse myocardial disease. The P-R interval at 0.20 suggested impending first degree auriculoventricular block. The diagnosis on discharge was arteriosclerotic heart disease and angina pectoris without coronary artery occlusion.

No similar episode occurred in the interval between February 1946, and October 1950, when the aforementioned abdominal symptoms occurred, and the patient was brought to the hospital for emergency treatment on the service of Dr. Francis Foran.

At autopsy, performed immediately after death, the following observations were made: The abdominal cavity contained 50 cc. of fresh blood from minute tears in the peritoneum near the left kidney. There was hemorrhage beneath the mesosigmoid close to its attachment to the posterior wall, and the blood extended laterally to the outermost edge of the left rectus muscle. The hemorrhage also extended to the region of the left kidney retroperitoneally and downward beneath the covering of the urinary bladder. In the region of the lower pole of the left kidney the hemorrhage extended to the right of the midline, across the duodenum, and into the mesentery for the small bowel for 8 cm.

The left adrenal gland was extensively dissected by hemorrhage, and some parts of the cortex resembled canary yellow islands of tissue in a sea of blood. The first impression was of an

adrenal tumor in which a blasting type of hemorrhage had occurred. The origin of this huge extravasation was, however, in a saccular aneurysm of the aorta just proximal to the bifurcation. The aorta was dilated to a circumference of 12 cm. at this point, whereas above the dilatation the greatest circumference was 7.2 cm. There was dissection under the lining of the aneurysm in a place 6 cm. in diameter and through an atheromatous patch 8 mm. thick. The splitting of the aortic layers proceeded circumferentially to a rupture through the adventitia and produced the hemorrhage already mentioned. The free blood in the retroperitoneal fat amounted to 1,500 cc. There was, in addition, at least 500 cc. of blood in the aortic wall, in the mesentery for the small bowel, and in the fat close to the left kidney.

The lungs were emphysematous anteriorly and subcrepitant dorsally. There were coal-pigmented nodules with gray fibrous centers, resembling arrested tubercles in the upper lobe and apex of the left lung. There was a similar nodule in the lower lobe of the right lung.

The root of the aorta had its lining altered about fifty percent by fatty and fibrous patches. This change increased gradually throughout the thoracic and

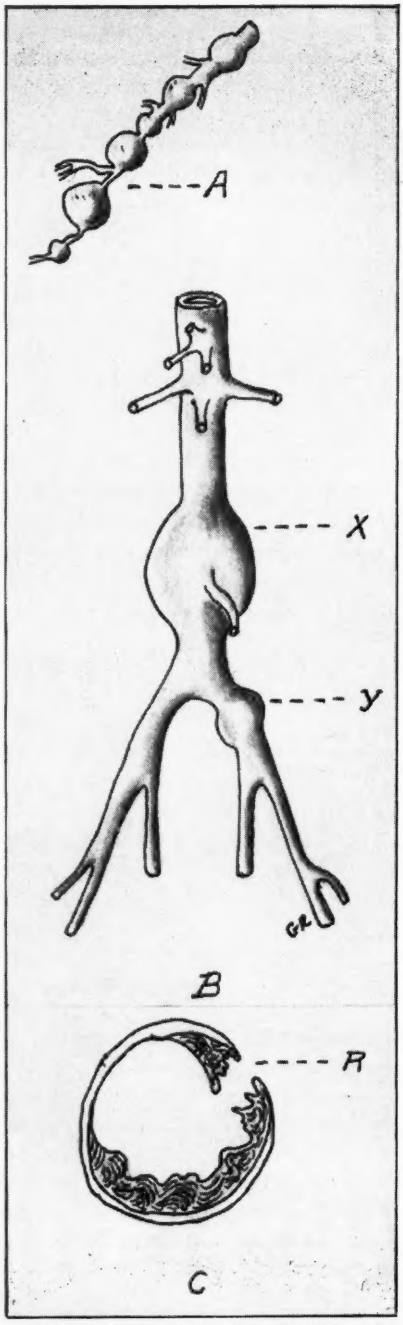


Figure 1

Illustrating at (A) the six aneurysms of the posterior coronary artery. (B) The abdominal aorta with an aneurysm at (X) and the left common iliac artery with an aneurysm at (Y). (C) A schematic representation of the aortic aneurysm to indicate the extent of dissection within the wall at the middle of the rupture site (R).

abdominal portions of the aorta, so that at least eighty-five percent of the lining was involved at the bifurcation and in the common iliac arteries.

The heart weighed 465 grams. The outer wall of the left ventricle was 27 mm. thick, while that of the right ventricle was 5 mm. In many places 3 mm. of the latter measurement was due to fat. The circumferences of the heart valve rings were as follows: tricuspid 13.2 cm., pulmonic 7.4 cm., mitral 9.6 cm., and aortic 7.8 cm.

The coronary arteries were markedly tortuous, and the lining of the left was altered about 65% by fatty patches. The lumen was 11 mm. in circumference close to the mouth and was irregular but nowhere occluded. The right coronary artery was phenomenally altered. The mouth was 5 mm. in diameter. There were six aneurysms of this artery, all along the main descending branch. (See Figure 1). The first dilation had a calcified lining and was 8 mm. long and 11 mm. in diameter. It began 18 mm. from the artery mouth. The second aneurysm was centered 25 mm. from the first, was of the same size, and had about two-thirds of its lining covered with a partially organized blood clot. The third aneurysm was pouch-like, 6 mm. in diameter, and involved the outer aspect of the artery and almost all of the lining. With the exception of 10% of the left edge, the lining was covered with a stiff red clot, 1 mm. thick. The next aneurysm began 2 mm. more distally, was 18 mm. across, and had a clot obstructing both its entire lumen and that of the vessel on which it was situated. The clot was rubbery and dark red.

After a sclerotic course of 7 mm., the artery expanded into an aneurysm 2 cm. in diameter. This was filled with gray to pink grumous material, which resembled disintegrated clot with added fatty matter from an opened atheromatous region. After a straight interval of 5 mm., the last aneurysm was discovered. It was 7 mm. in diameter, and its lumen was filled with fresh red blood clots.

The myocardium of the apex of the left ventricle was scarred in one place 15 mm. in diameter. The lining of the left ventricle on its septal aspect contained eight white scars from 1 to 30 mm. broad and 7 to 12 mm. long. The outer wall of the left ventricle was extensively scarred by white patches from 1 to 5 mm. in diameter, especially in the central third.

There was also an aneurysmal dilatation, 15 mm. in diameter, of the left common iliac artery 1 cm. distad to its mouth. The wall of the sac was sclerotic and as much as 7 mm. thick. The lining was partially dissected away by blood beneath it in a region 12 mm. in diameter. Here the detached lining was wrinkled. The other organs were remarkably free of changes other than sclerosis of the arteries. The spleen weighed only 75 gm., and its pulp was coarse and nearly bloodless.

Microscopic examination disclosed the following changes: The heart muscle cells showed hypertrophy and had moderate accumulations of polar liopochrome pigmentation. The scar tissue was, in general, old, but occasional sparse lymphocytic infiltrations were seen near the edges of the scars. There were also patches showing imperfect staining and haziness as is found in very early myocardial infarction.

Sections from the aortic aneurysm, whose dissection and rupture caused

death, showed partial calcification in the atheromatous patch through which rupture occurred. The media showed the irregular track of dissection from the lining to the surrounding fat.

The aneurysm of the left common iliac artery showed a clot attached to the lining on its outer aspect. There was fresh hemorrhage into all layers of the artery wall. There was advanced sclerosis with partial calcification of the lining.

Sections of the aneurysms of the right coronary artery showed the changes typical of advanced senile sclerosis. There was extensive hyalinization of the media of the third aneurysm and acicular vacuolation indicative of cholesterol deposition. There was partial calcification of the lining.

The left adrenal gland had abundant hemorrhage into both cortex and medulla and into the surrounding fat. The cortical portions of the gland were dissected away from the capsule by hemorrhage in many places. In other regions there was indentation of the capsule from hemorrhage in the surrounding fat.

The prostate gland was involved in a benign hyperplasia of the left lateral lobe. This contained several well-circumscribed adenomas, in which there was papillary overgrowth of lining cells. There was a slight interstitial lymphocytic infiltration close to the adenomas.

The anatomic diagnosis summarized the findings as follows: marked generalized senile arteriosclerosis; dissecting aneurysm of the abdominal part of the aorta; extensive hemorrhage into the retroperitoneal fat, urinary bladder wall, left adrenal gland, the mesentery for the small bowel; six aneurysms of the posterior coronary artery (several thrombosed); aneurysm with early dissection and clot formation of the left common iliac artery; benign prostatic hyperplasia; and scars of the knees (repair of quadriceps femoris tendons).

Further detail concerning aneurysms of the coronary arteries with especial reference to this patient is available in an additional article.[1]

Case II—The second patient, H. P., was a mechanical engineer 71 years of age who had never experienced a serious illness until August of 1950. Then he developed a cerebral thrombosis and became involved in a minor automobile accident. While in the hospital he became disoriented and developed an alarming anxiety. In this state he took a huge dose of sleeping tablets with suicidal intent. After a coma of one day he made a rapid recovery and was able to resume his occupation.

On October 19, 1951, he noted a peculiar bloated sensation in his lower abdomen and this disappeared in a short time. This sensation returned in the early morning of October 20, and the patient drank one-half bottle of magnesium citrate. After three watery bowel movements he felt better and went to work. In the late afternoon, the indefinite abdominal sensations returned and were quickly succeeded by agonizing pain in the left lower quadrant and entire lumbar region. He was taken home in a fellow-worker's automobile, moaning with pain and hugging his knees to his abdomen. The family physician, fifteen minutes later, found him writhing on the floor with his knees drawn up and his cries audible

[1]Multiple aneurysms of the right coronary artery. Death from a ruptured aneurysm of the abdominal aorta. G. J. Rukstinat, J.A.M.A. 149:1129, 1952.

outside of the house. The pulse was 50 to 60 per minute and the heart tones were faint and regular. The patient was brought to Loretto Hospital by ambulance at 6:45 p.m., October 20.

Medical and surgical consultants found severe tenderness and moderate rigidity of the left lower abdominal quadrant. The rectum was empty. Although tentative diagnoses of coronary occlusion and mesenteric thrombosis were made, the surgeon thought immediate operation imperative. At the laparotomy, a retroperitoneal hemorrhage was found from the midline to the left lateral peritoneal reflection. The blood extended from the level of the left renal artery to the pelvis. The diagnosis of dissecting aneurysm of the abdominal aorta was made and the patient's abdomen was closed.

An electrocardiogram made on October 20, was interpreted as follows: "Rate 90. Rhythm-auricular fibrillation. Q.R.S. 0.09 seconds (normal) inverted in I; diphasic in II and upright in III. T waves inverted in I; upright but low in II and III. Unipolar precordial leads—diminutive r and broad s in V-1-2-3-4. Q.S. in V-5 and 6. T waves flat in V-4-5 and 6. Unipolar extremity leads—Rs and flat T waves in A.V.R. Q.S. and inverted T waves in A.V.L. with slightly elevated S-T sectors. Rs with low T waves and depressed S-T sectors in A.V.F. Conclusion—The pattern closely resembles that seen in dextracardia. However, there is sufficient evidence of myocardial abnormality to assume that the changes are on the basis of a large infarction of the lateral wall of the left ventricle."

The patient lived until 10:45 p.m., a hospital stay of 28 hours. The additional dissection of the aortic wall and hemorrhage retroperitoneally are obvious from the following excerpts from the necropsy record.

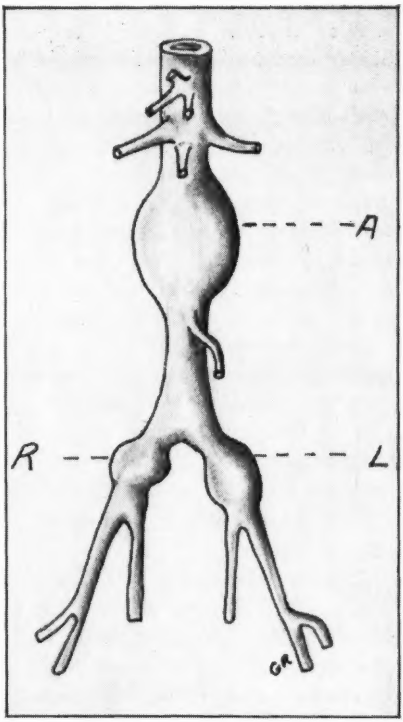


Figure 2

Illustrating at (A) the aneurysm of the abdominal part of the aorta and at (R) and (L) respectively the aneurysms of the common iliac arteries.

The lining of the root of the aorta is changed at least fifty percent by fatty patches and these occur in conglomerate masses at times. The aorta cusps are calcified and stiffened. The back portion of each leaflet contains a series of irregular yellow nodules, some of which also occur in the lining of the sinuses of Valsalva. The lining of the anterior coronary artery is altered at least fifty percent by fatty patches in

general, but in the proximal 4 cm. the sclerosis involves at least seventy-five percent of the lining. There are fibrous adhesions between the omentum and a small hernial sac on the right side and the omentum is grown into the sac for about 3 cm. There is a huge retroperitoneal hemorrhage, especially on the left side. This extends into the mesentery for the small bowel and pushes the mesentery upward from the spine for about 5 cm. The hemorrhage has dissected beneath the peritoneum on the left side of the abdomen and down into the upper part of the inguinal canal. It also has dissected the mesentery for the sigmoid part of the colon. Much of the blood is clotted. Some of it is still liquid. The hemorrhage extends up to the diaphragm.

There is a huge aneurysm of the abdominal part of the aorta (See Figure 2) beginning 2 cm. distal to the renal arteries and the sac of the aneurysm is 12 cm. long, bulges dorsally especially, and on the left side and is filled with a laminated clot, most of which, on the internal aspect is ridged. The rest of it is crumbly and pink to brown. Recent thrombi as much as 8 mm. in diameter cling to the inner surface. Dissection from this aneurysm through the aortic wall has taken place anteriorly and especially to the left of the vertebral column. However, there is blood free beneath the peritoneum on the right side also. This extends downward along the right common iliac artery. Each common iliac artery is involved by an aneurysm and on the left side this aneurysm is about 5.5 cm. long and 3.5 cm. in diameter. It is filled with a firm organized clot. The aneurysm on the right side has partially dissected through the wall which contains several calcified plaques which are contiguous except for a few small cracks between them.

The free blood retroperitoneally is estimated at 1500 cc. The liver is moderately fatty and its lobules are indistinct. There is no noteworthy change of the lining of the gallbladder or extrahepatic bile ducts.

The anatomic diagnoses were as follows: marked generalized senile arteriosclerosis; huge saccular aneurysm of the abdominal part of the aorta with recent rupture; saccular aneurysms of both common iliac arteries; huge retroperitoneal hematoma; exsanguination of the body; fatty changes of the liver and pancreas; fibrous myocarditis; and fibrous adhesions between omentum and right inguinal hernial sac.

Case III—J. P., a barber, 64 years of age had had a gastrojejunostomy for gastric ulcers in 1933. In 1935 he developed a duodenal ulcer and for this he had received medical treatment in succeeding years. For ten months prior to his final admission, he had had polyuria and incontinence. Nocturia occurred as often as ten times per night.

On December 2, 1952, he had a dull ache in the right lumbar region. This later radiated to the right inguinal region and thigh. During the next two days, pain of an acute distending nature was experienced in both upper and lower quadrants on the right side. Vomiting followed the onset of the pain. For the next two days the pain varied in intensity but often subsided to a slight lumbar ache. The additional complaints of anuria were present and a mass in the right side of the abdomen.

The patient was hospitalized on December 6, 1952, with a diagnosis of right renal colic and retroperitoneal

tumor. He was sweating and clammy and had a blood pressure of 108/80, a pulse of 120 which was weak and irregular and respirations of twenty per minute. His abdomen was distended and rigid, especially in the right upper quadrant. When the patient attempted to sit up, the pain was excruciating and then seemed most intense in the right kidney region.

The patient was cystoscoped and the urinary bladder was found to be trabeculated. The middle and lateral lobes of the prostate gland were enlarged slightly. Clear urine was obtained on catheterizing the left ureter. On the right side, bloody urine was obtained after a number six catheter was passed above an apparent obstruction. The impression was gained of an intrarenal hematoma of the right side. The possible existence of a renal neoplasm prompted a decision to explore the right kidney region.

A right lumbar approach revealed a huge extraperitoneal hemorrhage containing organizing clots and fresh blood. The right ureter was deviated medially. When the clots were evacuated a red pulsating mass was seen medial to the kidney. Blood was spurting from an opening 2 mm. in diameter in this mass. The entire operative site was flooded with blood. Despite all efforts at packing the bleeding point and even after administration of five pints of compatible blood, patient died two minutes after operative wound was closed.

The conclusions of the roentgenologist are enlightening in this patient. Following is an excerpt from the x-ray summary. Marked displacement of right kidney and ureter to the right anteriorly. This finding suggests some form of retroperitoneal mass. It could represent neoplasm or inflammatory process. Serious consideration should be given to possibility of dissecting aneurysm in view of demonstration of calcification in the abdominal aorta along with the displacement of kidney and ureter. Left upper urinary tract not remarkable except for possible demonstration of at least one cyst involving lower pole of left kidney producing separation between some of the prominent caliceal structures. Prostatic hypertrophy and cystitis.

The autopsy revealed the following important conditions. The lining of the peritoneal cavity is irregularly roughened by fibrous adhesions between the omentum and the region of the right inguinal hernial ring. There are also adhesions close to the midline of the abdomen above the navel where there is a thin laparotomy scar about 12 cm. long. There is hemorrhage beneath the peritoneum of the pelvis especially on the right side, also on the left and the front of the urinary bladder. The thickness of the hemorrhage is as much as 2 cm. This blood has dissected the peritoneum away from the underlying fat. It has also dissected into the mesocolon in the vicinity of the cecum into the root of the mesentery for the small bowel even to the region of insertion of the mesentery into the bowel. The hemorrhage also extends into the mesocolon and into the vicinity of the gallbladder. There is huge gauze packing retroperitoneally on the right. This is in a mass about 18 cm. vertically and about 12 to 14 cm. in diameter. It connects with a drain noted protruding from the upper angle of the recent surgical wound.

The abdominal part of the aorta is involved in a huge aneurysm which, by

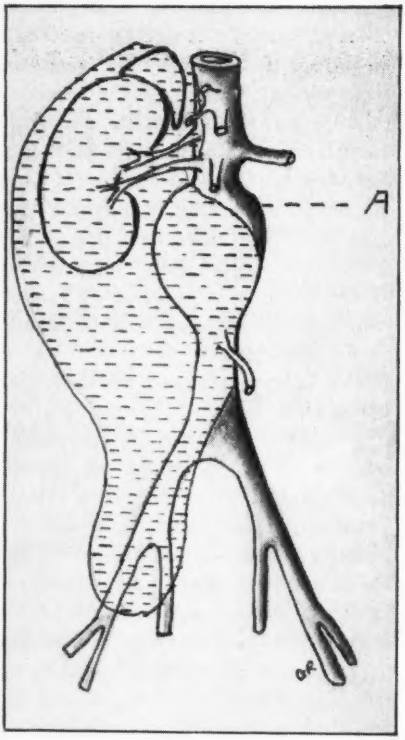


Figure 3

Illustrating at (A) the aneurysm of the abdominal part of the aorta. The shaded region represents the zone of major hemorrhage from the aneurysm.

actual measurement, is 9.5 cm. vertically, and circumferentially has a maximum width of 11.5 cm. There is dissection of the wall of the aneurysm from a rupture in its lining at the upper right edge. From this region dissection has taken place circumferentially from 4 to 9 cm. in width. The blood has broken out of the wall in the vicinity of the right suprarenal gland and kidney producing a huge retroperitoneal hematoma. The hemorrhage has dissected around the right suprarenal gland and around the kidney separating its fatty from its fibrous capsule. There are two arteries to the right kidney and the upper one is the larger. These arteries parallel each other at a distance of 9 mm. from the region of the aortic wall and 18 mm. from their insertion at the kidney. The right kidney shows an extensive region of depression which is about 8 cm. by 3 cm. in its greatest measurement and depressed 2 to 3 mm. below the regular contour of the kidney. This region resembles a zone of arteriosclerotic atrophy. There is only one artery to the left kidney and this kidney is markedly altered by retention cysts. In the upper pole there is a tense cyst 8 cm. in diameter and in the lower pole there is one 4 cm. in diameter. The left kidney with its unopened cysts weighs 380 grams.

There are dense fibrous adhesions between the gallbladder and the undersurface of the liver. There is a scar which deforms the pyloric region of the stomach, puckers it on its anterior and inferior surfaces. The circumference of the pyloric ring at the puckered site is only 2.1 cm. There is a posterior gastrojejunostomy with a double stoma. Each stoma fits two fingers easily. The lining of the urinary bladder is slightly edematous but otherwise unaltered. The prostate gland is of normal size.

The anatomic diagnoses are as follows: Marked generalized senile arteriosclerosis; dissecting aneurysm of the abdominal part of the aorta; extensive hemorrhage into the retroperitoneal region; tamponade of the kidneys, suprarenal glands and urinary bladder; eccentric cardiac hypertrophy; extensive scarring of both kidneys; huge cysts of the left kidney; disseminated fibrous myocarditis; scar of the pylorus

of stomach with marked narrowing of the orifice; old gastrojejunostomy; and recent nephrectomy incision.

Case IV—J. L., a retired clerk seventy-six years of age complained of belching, vomiting and "feeling full of gas" for three days. He had had no bowel movements during this time. The morning before admission he felt pain in his left upper abdominal quadrant. This became progressively worse during the day and persisted for twenty-four hours. During this period he passed "red" urine once but never had radiation of pain to the genitals. The admission diagnosis was possible bowel obstruction.

Soon after admission to the hospital the patient fainted while in the toilet. After being put to bed, he was in shock and remained unconscious. His blood pressure dropped to 60/40 mm. of Hq. and his heart sounds were inaudible. With the return of consciousness, he became restless and was examined with difficulty because of abdominal pain.

The x-ray conclusions the day after admission were possible dissecting aneurysm of the abdominal aorta; remote possibility of an echinoccus cyst within retroperitoneal space to be considered: and incidental degenerative arthritis of the dorso-lumbar spine.

There was evidence of serious circulatory impairment in the electrocardiogram also. "There is evidence of active myocardial injury such as would be produced by ischemia either absolute or relative. There is right bundle branch block such as might be produced by an infarct involving the interventricular septum."

During the thirty-six hours this patient was in the hospital, he complained of abdominal pain which varied from dull to sharp and severe. After the initial episode of coma, he remained conscious until death. The blood pressure returned to 110/80 at several readings and cyanosis was considerably relieved. The necessary preparations for an exploratory operation for bowel obstruction were under way when the patient suddenly died.

At autopsy the following was found. The lining of the peritoneal cavity is hemolysis stained and the cavity is filled with blood. This blood has its origin in the retroperitoneal region at the level of the insertion of the diaphragm and on the left side. When traced to its source, the blood is found to emanate from a tear in the aortic adventitia. The blood has dissected along most of the thoracic part of the aorta from a recent rent in the lining of an aneurysm just below the lower level of the arch. The dissection in the wall is for a distance of 17 cm. and in the upper part of the tract the blood surrounds the aorta in the media for about one-half of its extent on the right side. As the dissection progresses downward, more of the aorta is encircled until there is total encirclement at the upper level of the diaphragm. There is clotted blood behind the pleura on each side and this is in a mass from 1 to 12 mm. thick. It unites with blood clots caused by extravasation into the diaphragm and the perirenal fat on the left side. The blood has broken through the peritoneum dorsal to the stomach. It has flooded the peritoneal cavity.

The lining of the root of the aorta is altered about 75% by fatty patches and about 30% of these are partially calcified. Near the region of the tear in the aortic intima there are two sclerotic patches, each about 15 mm. across. At

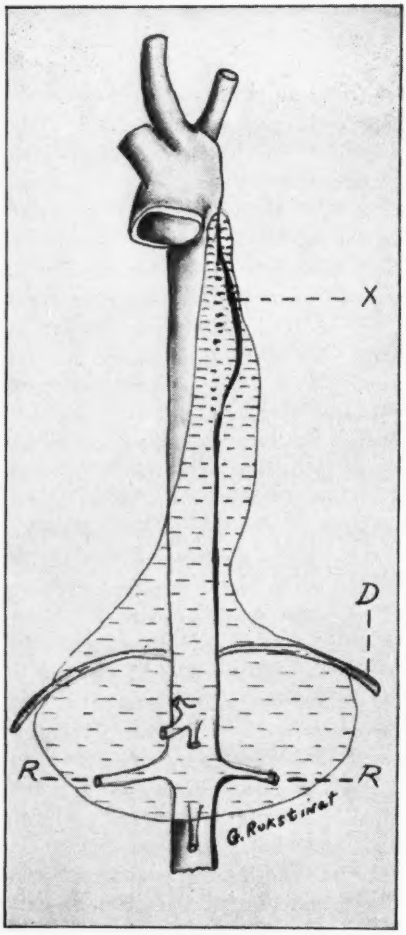


Figure 4

Illustrating the extent of the major extravasation (shaded) from rupture of a fusiform aneurysm of the thoracic part of the aorta.
Aneurysm (X) Diaphragm (D)
Renal Arteries (R)

the edge of one of these tears the blood has entered the aortic wall. Blood has dissected partially under the second patch. The maximum circumference of the aorta at the level of the intimal "break-through" is 15 cm. At the upper edge of the aneurysmal bulge, the circumference is 9 cm. The aneurysm is fusiform and has a slight left-sided prominence. (See Figure 4.)

Each lung weighs about 750 grams and is partially atelectatic. There is blood free beneath the pleura of each lung close to the hylus in a layer 1 to 2 mm. thick and extending peripherally for as much as 8 cm. from the root of the lung.

The heart weighs about 385 grams and its outer wall of the left ventricle has a maximum thickness of 24 mm. There are occasional patches of white scar tissue scattered throughout the interventricular septum and the outer wall of the left ventricle. The heart valve rings are within normal range. There is extensive calcification of the aortic cusps at their insertions and of the anterior mitral leaflet throughout about two-thirds of its extent. The coronary arteries are tortuous and sclerotic but unobstructed.

The other changes in the body were compatible with the age of the patient and the recent extensive hemorrhages. The histologic picture in the aorta was that of atherosclerosis both within the aneurysm boundaries and at various other levels. The disseminated fibrous change in the myocardium was in keeping with the degree of sclerosis of the coronary arteries.

The changes in the aortas of these four patients were of the type usually designated senile sclerosis or atherosclerosis. In no instance were there alterations compatible with the condition known as medionecrosis aortae idiopathica cystica, #1 and #2.

(To be concluded next month.)

Office Management of A Proctologic Patient

LOUIS B. GOLDMAN, M.D.
Chicago, Illinois

The office proctological examination should be carried out in such a manner that the patient will have complete confidence in the examining physician, and have ease of mind before, during, and after the consultation.

We must all be mindful of the fact that the patient does not come to the physician's office for a routine rectal examination. He comes because he has symptoms relating to his rectum, or because he fears that dreaded term "Cancer."

It is the duty of every physician to take an ano-rectal history on every adult, and to at least do a digital examination on every patient who has symptoms relating to his anus, rectum, or colon. Only in this way will he be able to diagnose ano-rectal pathology in its infancy, and reduce the morbidity and mortality.

The purpose of this paper is to show how a proctologist conducts an office consultation.

The office should be clean and well ventilated, soiled linens and paper should be kept in air-tight containers. All long instruments should be kept out of view. There is absolutely no reason why an office where rectal examinations and treatments are being carried out should have different odors than any other medical office.

At the first consultation the patient is seated in a comfortable chair facing the examining physician and given a few minutes to compose herself. A careful history is then taken.

The first question usually asked is "What is your trouble?" The patient usually replies, "Piles," "bleeding piles," or "itching piles." These terms cover a multitude of sins. The examiner then asks leading questions. The first question asked is:

Bleeding

a. Is it associated with bowel movements?
b. Amount, one must remember that a few drops of blood will color a toilet bowl full of water.
c. Duration?
d. Is it associated with pain?

Pain

a. Location, is it located around the edge or higher up?
b. Is the pain associated with bowel movements, and how long does it last?

Presented at the Sixth Annual Teaching Seminar, International Academy of Proctology, Chicago, Illinois, 1954.

c. Is it accompanied by bleeding?
d. Is the pain increasing in severity?
e. Character of pain; is it throbbing, lancinating or a dull ache?
f. Is the pain of long or short duration?

Protrusion

a. Does anything protrude from the anus? Does it protrude all of the time? Can it be pushed back? Is it painful, does it bleed? Is there a swelling around the anus?

Itching

Is it of long or short duration? Is it more pronounced at any time of the day or night? Is it constant or intermittent? What has been done for relief?

Discharge

Is it from the anus or around the anus?

Is it constant or intermittent?

Is it bloody?

Is it associated with bowel movements?

Bowel Movements

Has there been any change in bowel habits?

Is constipation present?

Does the patient have diarrhea?

Is it bloody?

How many stools a day?

Is it of long or short duration?

Are abdominal cramps present?

Does it follow the ingestion of any particular foods?

Stools

Is the stool of normal brown color?

Does it contain visible pus, mucus or blood?

Is there difficulty in evacuation?

General

Has patient had previous ano-rectal surgery?

Has patient had any rectal treatments, injection, etc.?

Has patient had an abscess?

Has patient had previous episodes of diarrhea?

Has patient had previous ano-rectal examination?

Has patient had previous x-rays?

Family History

Is there a history of tuberculosis, carcinoma, or polyps in the family?

The Examination The patient is placed in the left Sims position on a flat-top table with a clean towel under the left buttock. Another clean towel is placed over the right buttock. Thus the patient is properly draped. The examination, together with local anesthesia, popularized by Buie, begins.

Inspection As the buttocks are carefully retracted, the examiner says, "I am just looking at the outside of your rectum. I will be very careful not to cause you too much discomfort. I will not do anything to take you by surprise."

If the patient complains of pain one must not force the buttocks apart. If a painful lesion is present the examiner will be able to see or feel it if he will use patience, kindness and understanding.

One then looks for ulcerations, protrusions, new growths, openings of anal fistulas, and examines the character of the skin. If an opening of a fistula is present, one does not force a probe too deep, because this will cause severe pain, and the examiner may not be able to complete his examination. If a fissure is present, the examiner should not force his finger into the rectum, without first using some type of local anesthesia, either topical, or infiltration.

Palpation The perianal tissue is pal-

pated to determine if any masses exist and whether or not they are painful.

Digital Examination The well lubricated, cot-covered finger is then inserted into the anus. The patient is told, "I am now inserting my finger into your rectum. You will feel as though you are going to have a bowel movement. This feeling will disappear when I remove my finger." The entire circumference of the anus is palpated. The examiner notes the condition of the sphincters, and determines whether or not any masses are present. He then performs a bidigital examination. With the index finger in the rectum and the thumb on the outside, he palpates the perianal tissues for any areas of induration. The finger is then withdrawn, the finger-cot examined for gross blood, and this is confirmed by means of the Hematest tablet, which takes one minute.

Anoscopic Examination The warmed, well-lubricated anoscope, is then inserted into the rectum, and again the patient is told, "I am inserting an instrument into your rectum, and again you will feel as though your bowels will move. This will disappear when the instrument is withdrawn. After the anoscope is inserted the obturator is withdrawn and the ano-rectal area is examined for internal hemorrhoids, polyps, papillae (hypertrophied or normal), ulcerations, and condition of the crypts. A crypt hook explores the crypts. A crypt which admits a hook is said to be abnormal.

One also looks for the primary openings of fistulae. The entire circumference of the ano-rectum is examined, and the instrument is withdrawn, and the patient allowed to rest for a few minutes, while the examiner tells her of the sigmoidoscopic examination to make sure there is nothing higher up.

Sigmoidoscopic Examination This examination is carried out either on a tilt table or a flat-top table. If a flat-top table is used, the knee-shoulder position is the one of choice. The knees should be perpendicular to the table top, and the left shoulder should rest on a pillow, and the patient's head turned to the right. When using a tilt-table, the patient kneels on the knee-rest, and bends over on the table. He is then told that he will be tilted gradually and that he will not fall.

All instruments such as the 'scopes, light, suction tube, long swabs, biopsy forceps, fulguration electrodes, etc., should be close at hand and ready for use, but out of the patient's sight. There should not be any fumbling for instruments during the examination. The instruments should be handled carefully to avoid annoying metallic sounds and clashes.

The lights on the instrument should be tested before-hand to avoid embarrassment and confusion after the instrument is in the rectum. The anal canal is again well lubricated, the slightly warmed sigmoidoscope is held in the right hand for a right hand operator and the left hand for a left hand examiner. The thumb is kept on the obturator firmly until the scope passes the anal ring, it is then withdrawn and the light turned on. At this point air rushes in and there is a sensation of fullness. The patient is told that the rectum is clean and the sensation is due to air. The scope is then advanced under direct vision further into the rectum and following the lumen to the left or right, whichever the case may be. When the promontory of the

sacrum is reached, which is about 12-15 cm., the scope is angled anteriorly. The patient is told, "I am going around a bend and you will have a cramp in your abdomen. This will disappear when I remove the instrument. Please do not strain."

The scope then is passed into the rectosigmoid and the examination begins. Air inflation can now be used to balloon out the rectosigmoid beyond the scope. Air inflation is also valuable to separate a so-called "Sticky Bowel."

Some men use air inflation as an aid to introducing the sigmoidoscope.

As the instrument is withdrawn one notes the characteristics of the mucosa. One looks for ulcers, polyps, tumor masses, inflammatory changes. As the instrument is withdrawn and the rectal valves are reached, they are flattened out with the scope, because frequently polyps are found behind them.

As the instrument is being withdrawn the patient is told "I am withdrawing the instrument now. You will have the feeling of a bowel movement. Do not become alarmed."

As a general rule treatments are not started unless they are discussed with the patient previously. No remarks are made as to the findings during the examination. If the examination was unsuccessful the patient is asked to come back another day. If the patient was poorly prepared a Fleets Enema with one half ounce hydrogen peroxide can be administered with the patient in the knee-chest position. Satisfactory results usually follow in three to five minutes and the examination can be satisfactorily executed after.

The examination completed, the patient is helped off the table, given toilet tissue and allowed to dress. She then returns to the consultation room and is told of the findings and recommendations.

The first thing the patient usually wants to know is whether cancer is present. She must be told definitely if no cancer is present.

The next thing she will want to know is, is surgery indicated. We must be definite, there must be no hemming or hawing. She must be told yes or no when she leaves your office. She must be convinced. We must not temporize. We know that such conditions as tumors, fistulae, chronic fissures, prolapsing hemorrhoids, and polyps do not respond to medical management. Of course, if the patient absolutely refuses surgery, we are faced with two problems. The first is losing the patient, the second is treating the patient conservatively, but she must know that it is not the best form of treatment.

Any adult over thirty-five years of age who presents a proctological problem, especially those with bleeding, diarrhea, or change in bowel habits should have a barium enema and pneumocolon.

Summary

I have just reviewed what constitutes a good, adequate proctologic consultation and hope that it will stimulate more interest in colo-rectal examinations. Only in this way can we hope to reduce the mortality and morbidity caused by pathology in these areas.

7257 South Jeffrey Avenue

EDITORIAL

Address of Dr. Edward T. Whitney, in acceptance of the Honor Award Gold Medal and Citation for his eminence as a teacher of proctology, Seventh Annual Teaching Seminar, International Academy of Proctology, The Plaza, New York City, March 26, 1955.

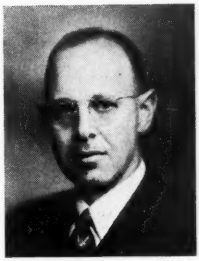
President Collins, Secretary Cantor, Officers of the International Academy of Proctology, Members and Guests—

I am deeply grateful to you for the honor you have rendered me tonight especially because it has to do with leadership in teaching.

Leadership is of two varieties. The first type is the natural born leader. Such a man sees a need, knows that he can fulfill that need and dedicates himself to the task of fulfilling it until he is successful in doing so. The second type has leadership thrust upon him. A need exists, he happens to be around, he accepts the consignment, and, measuring up, becomes successful in fulfilling it.

Your secretary is an example of the first type. He saw the need for an International Society in Proctology, he dedicated himself to the task of bringing it into being, he had the leadership to do it and this amazing, dynamic Academy is the result.

In my opinion, your officers should gather into a huddle, in the not too distant future, and decide to bestow upon *him* the honor which you have bestowed upon me tonight.



Dr. Whitney

I am an example of the second type. I had leadership thrust upon me. I had had three years of surgical interning way back in the days before the Boards became so popular. I was assisting a very busy surgeon both in his office and with his operating. He was surgeon-in-chief of the Boston Dispensary, and as such was the chief substitute most of the time, for the Chief of such a clinic is always responsible for the running off of the work even if no one else shows up to do it but himself. I was assistant to the chief-substitute. We were wholly responsible for the surgical clinic and had to fill in in all of the surgical specialties as well. When no one showed up in Surgical on the first floor, we substituted. When no one showed up on Gyn on the second floor, we went up there too. When there were patients to be seen in Urology up in the rarefied atmosphere of the third floor, and no

one came in to care for them, a call came down to us. It was the same way in Tumor and Vascular and Thoracic over in the new building or in G. U. down in the cellar and, last but not least, it was also true with Rectal, always bringing up in the rear, always at the bottom of the pile. As the result of all this we had to be semi-specialists in everything.

Finally, one day the Chief came to me and said, "Ed., you're it." "Well," I said, "I've been 'it' for some time but what is it this time?" "The Chief of Rectal is to retire and you are to take his place. Build that clinic up, will you?" I gulped hard, hunted up Rectal and, in addition to everything else that I had to do, I settled down to build it up as he had asked. It wasn't long before I became very enthusiastic and 'twas well that I did because soon a large Foundation came to us, wanting us to conduct classes in Proctology, among other things, to be given to some post-graduates who had been out practicing and were coming back for some refresher courses. We organized those classes. They must have been quite crude at first but, in due course, as we began to use visual aids such as lantern slides, movies, pathological and cadaver specimens, they improved and eventually became highly successful—at least, so we were told. Soon another Foundation asked us to do the same for them, and when these Foundations were not supplying us with students we ran courses on our own. Finally Tufts College Medical School came along and took us over.

The graduates of our courses number in the hundreds. Many of them have gone on and become full-fledged Proctologists. They are all over the world, several in South America, one in Spain, several in Canada and some in almost every state of the Union, and many are in this room tonight and I am proud of every one of them.

This afternoon someone asked me to tell you what I considered to be the true earmarks of a finished Proctologist. We believe we have the background to make such an appraisal for, in addition to teaching, we have to train men to operate our clinic, which at times runs fairly heavy.

Such a discussion will of necessity, require a few words which are not entirely delicate but we have all had our dinner and I am sure that the ladies, being married to Proctologists, are used to listening to their jargon at the dinner table. I am proud that I was able just now to remember to use the words dinner and dinner table instead of supper and supper table for I do not usually do it. I am a real down east Yankee of which there are but few left, and we have a vernacular all of our own. If we want to say, "taint so," we say "taint so" and if we want to say "twant so" we say "twant so." And instead of saying breakfast, lunch, dinner and then supper (at one A.M.) we say breakfast, dinner and supper. For us, supper *ends* the day; it does not *begin* it.

With that in mind, let me tell you what I consider to be the earmarks of a finished Proctologist. Firstly, when he is making his examination of a patient with a spastic fissure, he, one way or the other, is able to do so without sending every other patient in the building scurrying forth in abject terror. I'll admit it does help some to have a long, narrow, lanky, Yankee index finger such as I have! Then, in making the sigmoidoscopic examination, he has learned the art of taking one step back-

wards in order to take two steps forward. He doesn't try to push his way right through the sacral promontory, for it isn't any too elastic. I expect him to do nice clean-cut surgery in a hemorroidectomy as a routine, but do not consider him a finished product until he can handle himself expertly when faced with a very long, sinuous fistula with an obstructed internal opening which cannot be found and through which not even some dye will go, not to mention a probe. When I see him cleanly dissect out the whole tract, peeling it off the muscles and doing a nice plastic on that part of the sphincter through which he must cut, then,—I consider him a real Proctologist.

And now may I once again express my gratitude for the honor which you have bestowed upon me tonight.

Acknowledgements

The Executive Committee of the International Academy of Proctology and its members wish to thank Burton, Parsons & Company for sponsoring the cocktail party at the close of the Academy's Seventh Annual Teaching Seminar, March 26, 1955, at the Plaza Hotel. Needless to say, this was the perfect climax to a week of serious study and discussions, and was greatly enjoyed by all those who attended.

Also, the winners of the beautiful door prizes wish to thank the companies who so generously donated these prizes. The contributing companies were:

DESITIN CHEMICAL CO., manufacturer of Desitin Hemorrhoidal Suppositories with Cod Liver Oil.

FULLER PHARMACEUTICAL CO., manufacturers of Tucks.

ARLINGTON-FUNK LABS., a division of U. S. VITAMIN CORP., manufacturers of Panthoderm Cream.

MAX WOCHER & SON COMPANY, manufacturers of proctologic instruments.

ROMAINE PIERSON PUBLISHERS CO., publishers of THE AMERICAN JOURNAL OF PROCTOLOGY.

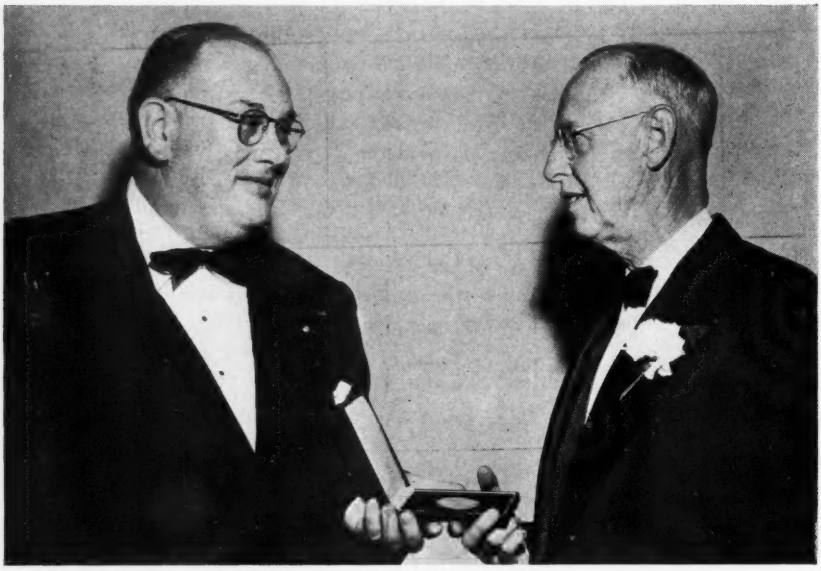
DOHO CHEMICAL COMPANY, manufacturers of Rectalgan.

Seventh Annual Convention

The Seventh Annual Convention of the International Academy of Proctology, the outstanding Proctologic teaching event of the year, convened at The Plaza, New York City, March 23, 24, 25 and 26, 1955. An unusually instructive program, covering both anorectal and colon disease, was well received by a very large audience.

Of particular note were the two panel presentations, one on anal, rectal and colon malignancy, and the other on ulcerative colitis.

The Jersey City Medical Center wet clinics, the morning of the 24th, and the lectures that afternoon, were both well attended, and were unusually instructive. Both of these sessions were under the guidance of the International Secretary General of the Academy and Surgical Director of the Jersey City Medical Center, Dr. Earl Halligan.



Dr. Donald C. Collins, President of the International Academy of Proctology (1954-1955) presents the Gold Medal Award of the International Academy of Proctology to Dr. Edward T. Whitney of Boston (right), for his distinguished achievements as a teacher of Proctology at Tufts University College of Medicine. This award, designating Dr. Whitney as a Master of Proctology, was accompanied by a hand illuminated and engrossed citation.

The award will be listed in the Blue Book of Awards (published by The A. N. Marquis Company, Marquis Publication Building, Chicago 11, Illinois, publisher of Who's Who, etc).

As has been the custom, members of the Medical profession, whether or not affiliated with the Academy, were cordially invited to attend the Convention sessions, without fee. The response, as usual, was overwhelming, demonstrating once again the need for teaching in proctology.

The Motion Picture Seminars were especially well attended. Indeed, this feature of the Annual Teaching Sessions has become of such increasing importance that two separate film sessions were presented at this meeting. At each of these sessions there were sections devoted to anorectal surgery, and sections devoted to colon surgery.

The Annual Banquet and Dinner Dance, as usual, was most successful. Honored guests at the banquet included Dr. Edward T. Whitney, surgeon in chief of the Rectal Clinic of the Boston Dispensary, New England Medical Center, and instructor in proctology at Tufts College Medical School. Dr. Whitney received a special Citation and Gold Medal Award from the Academy for his pioneer work in the teaching of proctology.

Dr. Alfred Angrist, Professor of Pathology at the Albert Einstein College of Medicine, New York City, accepted the first grant in the amount of $1,000.00, designated by the Academy for the development of a proctologic pathologic tissue slide library for teaching purposes at that college. This presentation was made by Alfred J. Cantor, M.D., Secretary of the International Academy of Proctology.

A second grant in a like amount was presented to Monsignor John McNulty, President of Seton Hall, designated by the International Academy of Proctology for the establishment of a chair in Proctology at the newly formed Seton Hall College of Medicine, in Jersey City. This is to be filled by eminent visiting Proctologists from all parts of the world. This presentation was made by Dr. Earl J. Halligan, International Secretary General of the Academy and Director of Surgery, Jersey City Medical Center.

Dr. Earl J. Halligan presenting check to Monseigneur John McNulty, (left).

Dr. Alfred J. Cantor presenting check to Dr. Alfred Angrist, (right).

Other honored guests at the Convention included Dr. Arkell Vaughn, Clinical Professor of Surgery, Loyola University, Stritch School of Medicine, and President of the Illinois State Medical Society, and Jose Iglesia De La Torre, Professor of Clinical Surgery, University of Havana Medical School.

The Treatment of Severe or Fulminating Ulcerative Colitis*

JULIAN M. RUFFIN, M.D.†
Durham, North Carolina

Chronic ulcerative colitis is a disease whose etiology is unknown and whose pathogenesis is poorly understood. It is not surprising, therefore, that its treatment has been non-specific and empirical. The long list of therapeutic agents which have been recommended in the past, only to be discarded later, is incontestable evidence of the inadequacy of any of these remedies.

Intelligent treatment of ulcerative colitis is dependent entirely upon a knowledge of the clinical course which these patients are likely to follow. The patient whose disease is mild responds in a most satisfactory manner to conventional medical therapy, whereas identical treatment may prove of little value in the severe or fulminating case. Failure to recognize the disease in its severe form may well result in an early fatality.

Clinical Course The clinical course of patients having chronic ulcerative colitis may be divided into three groups: the mild, the moderately severe to severe, and the fulminating. The degree of severity of the disease is determined by the number of stools per day, the presence or absence of constitutional symptoms, erythema nodosum, arthritis, and radiologic and proctoscopic findings. In one hundred consecutive cases recently analyzed at Duke Hospital, 33 were classified as being mild; 62 moderately severe to severe; and five fulminating. Those patients classified as having a mild colitis did well under conventional therapy, with 85% improved, or recovered. In the moderately severe to severe group, however, roughly 50% improved under medical treatment, while in the acute fulminating group, half of these patients died and the other half came to surgery.

In our experience the favorable factors affecting the course of the disease are: left-sided involvement only, mild initial severity, and clinical recovery

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* Author's Abstract from Modern Medicine, Feb. 1, 1955. From the Department of Medicine, Duke University School of Medicine, Durham, North Carolina.

†Professor of Medicine

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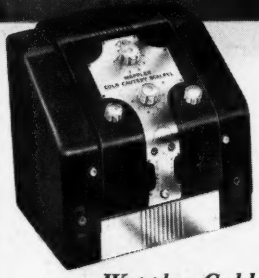

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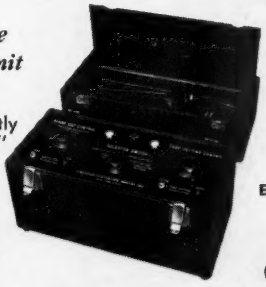

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between attacks. Such cases, in general, do well under conventional medical therapy. On the contrary, if one has an acute fulminating onset, right-sided disease, universal involvement, continuous symptoms without remission, the outlook is poor, and conventional treatment alone is unsatisfactory. Intensive medical therapy supplemented by ACTH or surgery usually will prove necessary.

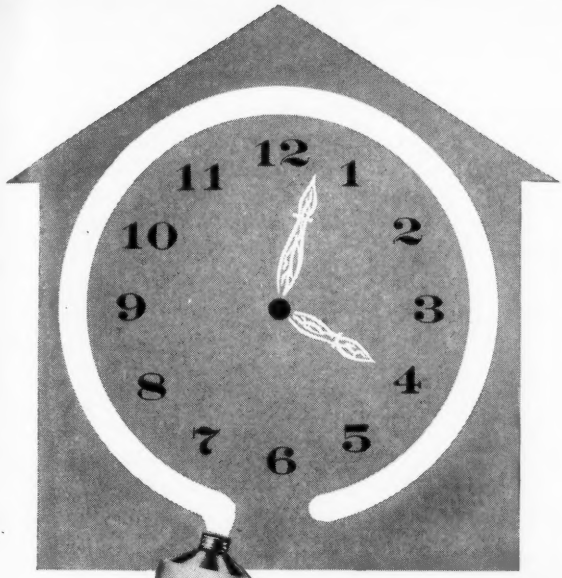
Conventional therapy includes: bed rest, preferably hospitalization, bland, low residue diet, supplemented by vitamins and liver extract parenterally. If the patient is anemic, iron or transfusions are indicated. Antispasmodics and sedatives also are advisable and psychotherapy in the form of encouragement, reassurance, and optimism is all important. A daily cod liver oil retention enema containing a non-absorbable sulfonamide is extremely valuable. The anticolinergic drugs have been reported to have a beneficial effect, but our experience does not confirm this observation.

The chemo-therapeutic and antibiotic agents consist of the sulfonamides, penicillin and streptomycin. The absorbable drugs are indicated when the patient is having constitutional symptoms. These drugs merely control secondary infection:—they do not cure the disease. Aureomycin and terramycin should be used with caution since severe ulcerative colitis has followed their administration.

Indications For Surgery The generally accepted indications for surgery are: medical failure, fulminating disease, uncontrollable hemorrhage, perforation, obstruction, fistula formation, and malignancy. It should be emphasized that the incidence of malignancy increases with the duration of the disease. It is a matter of importance, therefore, that the physician examine the patient proctoscopically and radiologically at frequent intervals. The one difficult question to answer is what constitutes a medical failure and when should one subject the patient to colectomy? There are no rules that can be made which are applicable to all and the decision as to operation has to be made on the merits of each individual patient.

Some years ago the operation of choice was ileostomy, to be followed by colectomy if necessary. The results of ileostomy alone, however, are so poor that this operation has been abandoned and it is generally accepted that once operation is decided upon, a simultaneous ileostomy and colectomy should be performed.

ACTH With the advent of ACTH, a useful and powerful therapeutic agent is now available. Its physiological effects are well known. The fever, arthralgia, and erythema nodosum, so commonly present in severe ulcerative colitis, subside in the most dramatic fashion following its use, usually within 24 hours after starting the hormone. The hormone suppresses inflammatory reaction, therefore it is not surprising that the diarrhea should subside and the proctoscopic appearance rapidly revert to normal. However, it alters fibroblastic activity and delays wound healing and giant or bizarre ulcers have been observed both proctoscopically and radiologically following its use. It should be pointed out that recovery following ACTH is a matter of days, whereas before the use of this hormone, a similar response would require weeks or months. It is important to realize



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2. Heimer, C. B., Grayzel, H. G., and Kramer, B.: Archives of Pediatrics 68:382, 1951.
3. Behrman, H. T., Combes, F. C., Bobroff, A., and Leviticus, R.: Ind. Med. & Surg. 18:512, 1949.
4. Turell, R.: New York St. J. M. 50:2282, 1950.

that a course of ACTH does not prevent recurrences.

In our experience the most satisfactory method of administration is to give 20 units in glucose, intravenously, over a period of 18-20 hours daily for seven days.

Thereafter the hormonal effect is tapered by a gradual reduction, over a three to five day period, in the length of administration of the drug. The value of oral cortisone over long periods of time is as yet to be determined.

The complications of ACTH are important and should be kept in mind whenever instituting this therapy. Hemorrhage or perforation of a peptic ulcer is well known. Perforation of the colon in patients with ulcerative colitis is certainly increased after ACTH and, of course, electrolyte im-balance must be carefully guarded against.

In summary, it should be emphasized that the cause of this disease is unknown, the clinical course is variable, and that there is no specific therapy.

Dr. Juan Nasio of Argentina (right), Editor of The International Book of Gastroenterology, presents Dr. Alfred J. Cantor, author of the section on protology in The International Book, with a citation for his pioneer work in proctology. This citation had been signed by eminent authorities in Spain, France, Germany, England and Argentina.

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SURGICAL TECHNIGRAMS. Edited by F. M. Al Akl, M.D. 338 pages. $12.00. New York, Toronto, London, 1954. McGraw-Hill Book Company.

This text, with its basic illustrations of surgical technique, is an excellent reference book for the surgical resident, and the occasional general practitioner surgeon. The book is devoted to technique exclusively. The illustrations are excellent line drawings, and show the step-by-step procedures in 36 of the most common operations. The stress throughout is on the anatomy and on the actual operative manipulation.

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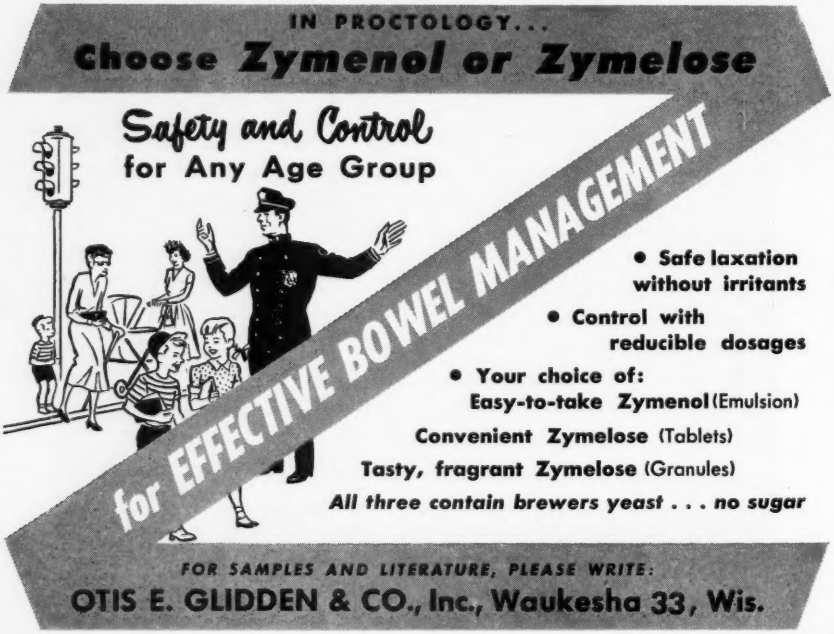
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